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TEXAS STATE JOURNAL OF MEDICINE

Volume 47 Number 5 Austin, Texas, May, 1951

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President's Page

"JOIN WITH ME"

Those speaking of Texas, whether within or without the state, usually deal in superlatives. Her vast size, her colorful history, her great achievements, even her great shortcomings always provoke stupendous adjectives. In similar vein the Medical Association of such a great state is big in numbers and deeds. The presidency of such an organization is likewise a big job and a great responsibility. In fact, the size of the task is enough to make even the stouthearted quail.

In being elevated to the leadership of this Association I am not unmindful of the great honor, but I realize the duties and responsibilities are in like proportion. The President is supposed not only to be the leader, but for his term, he is also the spokesman for his profession in his state. No man alone, however, can carry forward the policies of the organization. Only by the closest teamwork can we hope to achieve our objectives.

The continuity of effort in our organization is not broken by the advent of a new administration. Presidents, as well as other officers, come and go, but the long-range, constructive work moves on year after year, mapped and planned by the House of Delegates, the Board of Councilors, and the Board of Trustees. These units function as a closely knit whole whose problems, plans, and objectives are not changed abruptly by the advent of a new President. He only carries on the work already mapped out, adding as he is able his quota of assistance and effort to the program. Presidents come and go, new faces appear from time to time in the ranks, but the overall objectives and purposes are not thereby altered.

Like our national administration it seems that we must work in unsettled times. We move from crisis to crisis, often beset by forces from without which threaten not only to change or destroy our democratic way of life, but also to destroy the present system of medical service. Instead of having sufficient time for study and contemplation,

we must improvise from day to day to meet new threats and new crises. Such a state does not make for the best results in either government or medicine and naturally more errors are made under such stress. However, until more settled conditions obtain, we must move along this road. We must grapple with the new problems and new duties thrust upon us and not be overwhelmed by them. "We must take the current as it serves or lose our ventures." We must provide medical service of the best, while fighting all the time to preserve the present system of such service.

One great consolation of a democratic body, whether business, professional, or political is that its fortunes do not stand or fall as a result of the greatness or the foibles of any one man. They are arrived at through debate and deliberation, through conferences and compromises. The end result comes from the sincere thought and judgment of the majority. Such being the case it is reassuring to know that it is not my duty to "trust only to my own strength or in mine arm alone," but in these trying times to represent the combined thought and effort of the doctors of our great state. In the multitude of counselors there is safety.

That grand old man of medicine, Sir William Osler, teacher and philosopher, in bidding farewell to America insisted on the ideal of "life in water-tight compartments: to do the day's work well and take no thought for the morrow." Since "sufficient unto the day is the evil thereof" can we think of a better ideal at this time? In contemplating the coming year I remember Woodrow Wilson's invitation: "I ask all forward-looking men to join with me."

ALLEN T. STEWART, M. D.

Eighty-Sixth President, Texas Medical Association

LUBBOCK, TEXAS

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DR. ALLEN T. STEWART

Dr. Allen Thurman Stewart, Lubbock, is the eighty-fifth president who has served the medical profession in Texas and the first president of the newly named Texas Medical Association. He was installed at the concluding meeting of the annual session in Galveston May 2, 1951, having served as President-Elect since May, 1950.

Born in Sherman, Texas, on November 29, 1891, to John Wiley and Josephine (Mann) Stewart, Dr. Stewart was graduated from Sherman High School in 1908 and received a bachelor of arts degree in 1912 from Austin College in his hometown. After teaching school for five years, he attended the University of Texas School of Medicine in Galveston, where he received his doctor of medicine degree.

Dr. Stewart interned at St. Vincent's Charity Hospital, Cleveland, Ohio, in 1922 and 1923, and accepted a residency at Cleveland Maternity Hospital in 1923. He did postgraduate work at Washington University, St. Louis, and the University of Colorado, Denver. Since 1924 he has practiced in Lubbock, where he is now chairman of the board of the West Texas Hospital. He also is director of a loan company in Lubbock and of a life insurance company in Dallas.

Dr. Stewart's participation and leadership in the State Medical Association has included a term as president of the Lubbock-Crosby Counties Medical Society in 1933; Councilor of the Third District of the State Medical Association for the 1949-1950 term; and president of the Third District Medical Society for 1950-1951. Since 1946 he has been a member of the American Medical Association Committee on Rural Health, representing Texas, Oklahoma, and Kansas; he was delegate from Texas to the A.M.A. from 1948 until 1950 and has served on the Committee on Public Relations of the State Medical Association since 1946.

Since settling in Lubbock in 1924, Dr. Stewart has been engaged in general practice with emphasis on obstetrics. He is the author of several papers which have been presented before the State Medical Association and other

medical organizations and which have been published in the TEXAS STATE JOURNAL OF MEDICINE and elsewhere.

Dr. Stewart served as President of the Texas Association of Obstetricians and Gynecologists in 1945, and was certified by the American Academy of General Practice in March, 1948.

A member of the Methodist Church and of Alpha Kappa Kappa Medical Fraternity, Dr. Stewart is also a past president of the Lubbock Kiwanis Club with a record of nineteen years of perfect attendance; a member of the board of the Lubbock Independent School District for nine years; director of the Lubbock Red Cross Chapter; and former director of the Texas Technological Art Museum.

In addition to his capabilities in the field of medicine, Dr. Stewart received several honors during his college years for his oratorical ability. He was the recipient of the Austin College Scholarship Medal in 1910; winner of the San Jacinto Oratorical Contest the same year; winner of the Senior Oratorical Contest; representative of Austin College in the Texas Collegiate Oratorical Contest in 1912; and valedictorian of his college graduating class. In 1922 he was elected to membership in Alpha Omega Alpha, honorary medical fraternity.

On December 31, 1913, Dr. Stewart married Miss Anna Burke of Sherman. They are the parents of three children, Mrs. T. J. McWilliams, Denver, Colo.; Mrs. David B. Morrison, Lubbock; and Allen T. Stewart, Jr., student at Southwestern Medical College, Dallas.

Among his hobbies are hunting, fishing, and flying. Dr. Stewart has logged 500 flying hours in his personal plane.

Dr. Stewart's congenial personality, experience in his field, willingness to cooperate with other members of the Association in all matters for the interest of the medical profession and progress of the Association, as well as his ability as a public speaker are indications that his role as spokesman for the medical profession in Texas during 1951-1952 will be a successful one. His colleagues have faith in his leadership, knowing that Dr. Stewart displays the same pride in his office as President of the Texas Medical Association as he does in being known as "the family doctor."

REPORT FROM GALVESTON

Perhaps to the individual member of the Texas Medical Association, the most important action taken by the House of Delegates during the recent annual session in Galveston was to increase the dues to a total of $50. Because of unforeseen increases in the construction costs of the new Association building, now being erected in Austin, and a general advance in all operational costs, the Board of Trustees recommended that an additional $15 be collected annually from each member until the loan on the building is amortized, thus making the total state dues $50 annually beginning in 1952.

Probably the action most interesting to the public at large was the change of name from "State Medical Association of Texas" to "Texas Medical Association." The new name was proposed last year as an amendment to the Constitution and was approved this year. Proponents of the new name believe that there will be less likelihood of the organization being taken for a government agency and that the name is less cumbersome.

Decisions with far-reaching results were made with respect to postgraduate medical education. Upon the recommendation of the Council on Medical Education and Hospitals and after considerable debate, the House of Delegates voted (1) to underwrite postgraduate courses at the medical schools in Texas up to $2,500 each for the next twelve months' period, (2) to urge the medical schools to make their postgraduate courses self supporting if possible, (3) to disapprove the acceptance of personal remuneration by members of the Association from the State Health Department or other sources for attendance upon postgraduate courses, (4) to support the University of Texas Medical Branch

in Galveston, the Southwestern Medical School of the University of Texas in Dallas, and the University of Texas Postgraduate School of Medicine with headquarters in Houston in their request for funds from the State Legislature, and (5) to direct the President to appoint a special committee to help correlate postgraduate medical education of all kinds in Texas and to supervise expenditure of Association funds earmarked for underwriting courses at the medical schools. It was the feeling of most members of the House that the Association should cooperate in the development of a sound, comprehensive program of postgraduate medical education in Texas but that it should not encourage subsidy of such a program by the federal government.

The internal organization of the Association was modified in several respects. The duties of the Secretary were redefined so as to make him primarily responsible for signing the official papers of the Association and seeing that proper records are kept. Most of the routine duties which heretofore have been the responsibility of the Secretary were assigned to a newly established office—Executive Secretary. This executive is appointed by the Board of Trustees and need not be a member of the Association. Tod Bates, who was appointed by the Board of Trustees on an interim basis last August to direct activities at the central office, was continued as Executive Secretary, and Dr. Sam N. Key, Austin, was elected Secretary. Dr. Truman C. Terrell, Fort Worth, retiring member of the Board of Trustees, was elected President-Elect by acclamation, and Dr. William M. Gambrell, Austin, outgoing President, was elected Trustee. Dr. L. C. Powell, Beaumont, was named Vice-President; Dr. Robert B. Homan, Jr., El Paso, was reelected Speaker of the House of Delegates; and Dr. Hobart O. Deaton, Fort Worth, was chosen as Vice-Speaker of the House of Delegates. The office of Vice-Speaker, although fully approved by members of the House at this session, cannot become a reality until final action on an amendment to the Constitution next year, but it was agreed that it would be wise to proceed immediately with election of a person to fill the office as soon as it is created. The membership of the Committee on Public Relations was increased from five to seven, and the name of the Council on Legislation was changed to "Council on Medical Jurisprudence."

The House of Delegates adopted some resolutions for submission to the House of Delegates of the American Medical Association. One recommends that the sole responsibility for the evaluation, standardization, and control of medical practice in hospitals be vested in the Council on Medical Education and Hospitals of the A.M.A. Another requests that the Board of Trustees of the A.M.A. reevaluate the position of the national organization regarding delinquent dues for 1950.

Dr. Leo J. Peters of Schulenburg was honored by being named General Practitioner of the Year, and Drs. L. H. Reeves of Fort Worth and Felix P. Miller of El Paso, who had been nominated last year for membership emeritus, were elected to that status.

Dr. Allen T. Stewart, Lubbock, succeeded to the presidency of the Association and Mrs. O. W. Robinson, Paris, will fill the same office for the Woman's Auxiliary during the coming year. Mrs. Robert F. Thompson, El Paso, was named President-Elect by the Auxiliary, which met concurrently with the Association. In accordance with the decision reached last year, the 1952 annual session will be held in Dallas the week of May 4, and Houston was chosen as host city for the 1953 session.

Additional information about the session just past, which drew a total of 2,034 persons to Galveston for the largest attendance ever recorded for an annual session in that city, will appear in the June and July issues of the JOURNAL. The complete transactions of the House of Delegates and general meetings of the Association will be published in June, and the Auxiliary transactions in July. Members of the Association and Auxiliary should take time to

read both sets of transactions. These reports will help them understand the efforts made by their officers and committees in the past year toward furthering the program of the medical profession in Texas. They also will explain fully the decisions which were reached for the implementation of that program in the year ahead.

A NEW FIFTY YEARS

This month the Texas Medical Association enters its second half century under a continuous state charter. The document issued by the State of Texas in 1901 has been renewed for another fifty years. Although the Association will celebrate its one hundredth anniversary in 1953, the early years were stormy, and the charters obtained in 1853 and 1889 soon lapsed because of changes in organization or failure of some officials to keep the charter in good standing. A brief history of the struggle which led to reorganization of the Association on a sound basis in 1901 appears in the Organization Section of this JOURNAL.

It is appropriate to think fondly of the pioneers who ministered to the sick in the early days of statehood and who occasionally met to argue their mutual problems heatedly. Likewise, tribute is due those doctors who, by sheer will power, early in the twentieth century brought order to the medical profession in Texas and launched the Texas Medical Association as it exists today.

More important than looking back, however, is planning for the future, aware of the multitudinous problems which modern civilization brings to each physician and his patients. The same courage, the same willingness to debate important issues, and the same readiness to act which stimulated leaders of the organization during the past fifty years must be evidenced in the entire membership today, if the next fifty years are to reflect equal progress toward achievement of the goals which the Association has set.

CURRENT EDITORIAL COMMENT

VOLUNTARY INSURANCE

The importance to Americans of voluntary hospital and medical care insurance cannot be exaggerated. The phenomenal growth of the voluntary system is evidence that such insurance is needed and that the people believe in the voluntary American way of doing business. The vast majority of our citizens desire the freedom of individual initiative and responsibility. They want no part of so-called government handouts.

Primarily, the program has provided great assistance to the wage earner and his family. The pooling of premium funds to insure against a certain risk has provided a security against major hospital and medical expenses which the average family would find difficult to meet in any other way. Of even greater importance is the fact that this prepaid system has created a means through which the self-respect of millions of people is elevated and upheld. Self-respect cannot be given to Americans by a paternalistic government; rather, it is that "something" which only the individual can provide for himself.

In this time of economic inflation, America's hospitals have been greatly aided by our insurance program. In most large voluntary hospitals from 40 to 50 per cent of the admissions are covered by some type of insurance. In many instances, payments from this source are the transfusions necessary to keep the hospitals alive. A healthy hospital system provides better care for more people.

The physician and the surgeon have been given a freedom of action through this insurance program. No longer need they be con-

This department of the JOURNAL *presents editorial comments on current items pertaining to the science, art, and practice of medicine, contributed by members of the Texas Medical Association and scientists closely associated with the medical profession of Texas. Invitation is hereby extended to any member of the Texas Medical Association to submit such discussions for this department. The discussions should not be more than 500 words in length.*

cerned about the insured person being able to receive adequate hospital and medical care. Often the lack of hospitalization insurance handicaps the doctor because the patient feels that he cannot avail himself of adequate modern facilities. The provision of the finest medical care is the ethical duty of every doctor. Thus voluntary insurance means much to the family doctor.

There have been some complaints by patients, doctors, and hospitals that coverage is not broad enough in many instances. It has been pointed out repeatedly that complete comprehensive coverage, even if desirable, would be too expensive for the family which really needs protection; that present competitive companies are constantly broadening their benefits as experience warrants; that it is essential that misuse and abuse of the insurance program by the insured, the doctor, or the hospital be avoided. Such misuse is detrimental to the entire system. Abuses cause premiums to rise, resulting in fewer families being financially able to afford coverage.

This system will grow and prosper so long as the doctor, the hospital, and the insured help it expand. Many more millions of Americans will provide their own security against illness, if premiums remain reasonable and as experience grants expansion of benefits. This is the profession's program. It must succeed!

ROBERT B. HOMAN, JR., M. D.
El Paso, Texas.

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ORIGINAL ARTICLES

CUTANEOUS REFLECTIONS OF INTERNAL DISEASES

Comments on Diagnosis and Treatment

J. L. PIPKIN, M.D., San Antonio, Texas

THE subject of cutaneous reflections of internal diseases has been exhaustively covered by various articles and monographs, and to reiterate in the usual catalog fashion would serve no useful purpose. Such recent volumes on this subject as those by Wiener[17] and Behrman[1] can be recommended. Citing a few of the easily discernible cutaneous earmarks of internal diseases, stressing their medical significance, and reemphasizing the importance of their early recognition might be worth while. Commenting on the methods of diagnosis and modern treatment procedures should add some practical significance to the presentation.

In many instances the cutaneous earmarks precede by months or years the customary symptomatology of the disease entity. By the early recognition of these herald hallmarks of internal diseases and a search for the etiologic factors, proper treatment might arrest or reverse the early pathologic changes.

Read before the joint meeting of the Sections on General Practice, Eye, Ear, Nose, and Throat, Public Health, and Pediatrics, State Medical Association of Texas, Annual Session, Fort Worth, May 4, 1950.

LESIONS FROM LIVER DISEASE

Wilson[18] nearly 100 years ago described the frequent association of cutaneous arterial spiders (vascular spiders) with alcoholic hepatic cirrhosis. This pulsating, vascular, spider-shaped hallmark of liver disease is produced by an excess of estrogen, which the diseased liver is unable to inactivate. This same high level of estrogen in the blood at times produces in the male gynecomastia, enlarged and pigmented nipples, and a loss of axillary and chest hair. Spider nevi, commonly located about the face, neck, and upper chest, frequently antedate by many years the symptoms and signs of liver disease.

Palmar erythema (liver palms) is often associated with damage of the liver. Xanthomas on the palms, particularly along the crevices and folds, are frequently a cutaneous reflection of a liver disorder.

DERMATOSES OF QUESTIONABLE ETIOLOGY

Such dermatoses as erythema nodosum, lupus erythematosus and sarcoidosis exemplify the intricacies

of many cutaneous problems and bring to mind the difficulties encountered in establishing the cause. Only on an etiologic basis can specific and rational treatment be instituted. Each of the aforementioned entities presents definite, distinct, and fairly constant cutaneous patterns; yet the cause varies from time to time in each disease. The only common denominator in the cause of the previously mentioned three diseases is that at times all are associated with tuberculous infection.

1. *Erythema Nodosum.*—This inflammatory dermatosis is characterized by red, tense, brawny nodules, most commonly located on the shins; however, at times they are noted on the upper extremities as well as on other parts of the body. They recur monotonously. The disease repeatedly presents a uniform syndrome regardless of the etiology. It probably represents an allergic type of reaction to various internal bacterial, fungus, and virus infections, as well as to drugs. More than twenty infections such as tuberculosis, streptococcic infections (especially in sore throat and respiratory diseases caused by the beta hemolytic type of organism), gonococcic sepsis, and Resenow's diphtheroids have been reported to produce the classic entity of erythema nodosum. It has been associated with syphilis, leprosy, lymphopathia venereum, rheumatic fever, ulcus molle, sarcoid of Boeck, chancroid, and ulcerative colitis. Fungus infections such as trichophytosis and coccidioidal disease at times reflect a similar allergic cutaneous picture. Attendant constitutional symptoms such as fever, malaise, loss of weight, weakness, pain in the muscle and bone, polyarthritis, and occasionally rheumatic heart disease indicate the general systemic nature of this disease.

Such an array of etiologic possibilities illustrates the necessity of an exhaustive search for the cause of this cutaneous reflection of an internal disease.

Since the addition of the sulfonamides, sulfones, antibiotic drugs, and vitamins to our therapeutic armamentarium, the underlying systemic disease can be treated specifically if the cause is known. With these specific remedies at hand, the determination of the cause in each instance is even more pressing. Robinson[14] has recently reported excellent results in the treatment of erythema nodosum with aureomycin. Such treatment should be specific when certain types of bacteria are the cause, but aureomycin would be of little value if this cutaneous manifestation were associated with coccidioidal disease or leprosy. The dramatic results obtained with Fuadin in the treatment of erythema nodosum associated with Hansen's disease, as reported by Wolcott,[20] should emphasize the importance of determining the underlying cause for effective treatment.

TABLE 1.—*Clinical Types of Lupus Erythematosus.*

Clinical Type	Lesions	Location	Systemic Involvement	Abnormal Laboratory Data
Chronic Discoid	Early— Erythematous, scaly disks or plaques	Face Ears Scalp	No general symptoms or Chronic tiredness and "below par" feeling	None
	Late— Gaping follicles Telangiectasis Atrophy	As in early	As in early	Slight increase in sedimentation rate
Disseminated Chronic	Sometimes discoid erythematous patches	Face Ears Scalp V of neck Chest Back Forearms	Malaise	Slight leukopenia
Subacute	Dusty red patches Erythematous and edematous plaques	As in disseminated chronic More generalized distribution	Transitory arthralgia Low grade fever Renal irritation	Albumin and casts in urine
Acute	As in subacute	Face V of neck Chest	Lymph node enlargement Arthritis Polyserositis	Leukopenia Anemia Increase of serum globulin
Acute febrile	Diffuse erythema Erysipeloid lesions Marked edema Multiform lesions Vesicular Bullous Purpuric Telangiectatic	Hands Toes Mucous membranes Generalized distribution	As in acute Gastrointestinal symptoms with hemorrhage Nephritis Toxic hepatitis and splenitis Septic type of fever Verrucous endocarditis	Blood in stools Albuminuria, casts and red blood cells in urine Very rapid sedimentation rate Severe leukopenia with lymphocytosis

2. *Lupus Erythematosus.*—Lupus erythematosus is a general pathologic process of unknown etiology accompanied by a protean symptomatology; as a rule it is reflected in the skin as variable but typical lesions. The cutaneous picture can vary from a solitary, chronic, discoid plaque with no systemic symptoms,

on the one hand, to a violent, acute, generalized, multiform eruption with lesions of the mucous membrane accompanied by fever and prostration which eventually terminates in death, at the other extreme. Between the chronic and acute forms are gradations of the cutaneous picture with varying associated systemic symptoms. As the dermal picture gets more acute, the constitutional symptoms become more marked. Regardless of the type, stage, or extent of the lesion, it is generally accepted that all are clinical variants of the same disease. Table 1 enumerates the clinical types of lupus erythamatosus (as differentiated by Wise[19]) and lists the lesions, locations, associated general symptoms, and laboratory data.

The location and distribution of the lesion and the fact that the eruption is subject to actinic insult make the diagnosis evident in the majority of instances. The clinical diagnosis can be substantiated by a histopathologic study of the lesion in 75 per cent of the cases, according to Ellis.[5]

The diagnosis of acute disseminated lupus erythematosus can be substantiated by demonstration of the so-called "L. E." cell as described by Hargraves.[8] He observed this cell in the sternal marrow, and later it was demonstrated in the peripheral blood of patients with acute disseminated lupus erythematosus. Moffatt, Barnes, and Weiss[9] induced the "L. E." cell in normal peripheral blood by adding venous blood or plasma from patients with acute disseminated lupus erythematosus.

In treatment, ultraviolet rays, either natural or emanating from therapeutic machines or mazda-fluorescent tubes, must be strictly avoided.

If the conventional methods of treatment of the chronic disease, such as bismuth, gold, Mapharsen, and liver therapy, fail, such newer preparations as vitamin E, both orally and intramuscularly, and the salts of para-aminobenzoic acid should be tried. Recent reports on these drugs are encouraging. Foci of infection should be sought and eliminated.

Treatment of acute forms of the disease consists of bed rest and general supportive measures, including blood transfusions and a judicious trial of the salts of para-aminobenzoic acid. As reported by Zarafonetis, Grekin, and Curtis, [21] this drug seems of distinct aid in decreasing the acuteness of the disease, reducing the fever and preparing the patient for more drastic treatment and for cautious removal of foci of infection.

3. *Sarcoidosis.*—Dermatologists have long considered sarcoid disease a generalized granuloma which almost invariably involves the lymph glands and at times other organs but which spares the skin in about one-third of the instances. Table 2 exemplifies the symptom complex of sarcoidosis, listing the organs involved, the frequency and type of lesions, and the associated laboratory findings.

In the clinical diagnosis of sarcoidosis, typical or suggestive cutaneous lesions are present in 60 per cent of patients. These dermal lesions, correlated with changes noted in other organs and the laboratory data, should confirm the diagnosis. If the cutaneous manifestations are not typical, a histologic examination should substantiate the clinical impression; this examination should be of value in 90 per cent of the biopsies taken.[5] If no dermal lesions are present, biopsy of an enlarged gland should aid in differentiating sarcoidosis from Hodgkin's disease, lymphosarcoma, or other lymphoblastomas. The Kveim test, with histologic study of the papule, will aid if the diagnosis is questionable.

TABLE 2.—*Symptom Complex of Sarcoidosis.*

Organs Involved	Frequency (%)	Type of Lesion	Associated Laboratory Findings
Lungs and bronchi	90 to 100	Infiltrate at root radiating to bases	
Lymph tissue	90	Enlarged lymph nodes Superficial and deep Mediastinal Bronchial	
Skin	60	Nodules Small—firm, sharply demarcated, waxy yellowish brown Large—same character Diffuse infiltrates	Blood chemistry Increase in Calcium (marked) Phosphorus (slight) Total protein Globulin fraction Nonprotein nitrogen Lowered or reversal of albumin to globulin ratio Decrease in urea clearance Increase in sedimentation rate Normal blood count except for occasional eosinophilia Urine normal
Eyes	40	"Mutton fat" deposits in iris and posterior part of cornea	
Bones	10	Reticulated and meshlike rarefaction of small bones of the extremities	
Any or all organs: Liver Spleen Kidneys Heart Gastrointestinal tract Central nervous system			

In all organs involved a sarcoid type of infiltrate is seen.

Calciferol therapy (vitamin D_2) has been advantageously used by Curtis[4] and is reputed to resolve rapidly the sarcoid infiltrates. This irradiated ergosterol is given in doses of 50,000 units three times a day. It must be remembered that large doses of calciferol during a long period often produce toxic symptoms, and the clinician should be alert for these manifestations. Favorable reports on dihydrotachysterol, 3.75 mg. for three days and 1.75 mg. thereafter, are on record.

In appraising any form of therapy in sarcoidosis, the erratic and varied course of the disease must be remembered. Commenting on this, O'Leary[12] stated that the evaluation of calciferol therapy is difficult since the lesions in 50 per cent of patients with the disease will resolve spontaneously within five years with no treatment. He further stated that chaulmoogra oil (Chaulmestrol), which has given consistently good results during the past fifteen years, should not be too hastily discarded.

LYMPHOBLASTOMA

The cutaneous reflections of the clinical entities grouped under the broad generic term of "lymphoblastoma" or "lymphoma," namely, mycosis fungoides, Hodgkin's disease, leukemia cutis, and lymphosarcoma, can be divided into two groups: (1) specific lesions, showing specific histopathologic changes and (2) nonspecific (toxic) lesions, showing mainly an inflammatory infiltrate with or without specific cells. Histologically and clinically, a sharp line of division between these two groups is not apparent in all instances; they may merge imperceptibly at times.

TABLE 3.—*Cutaneous Manifestations of the Four Forms of Lymphoblastoma.*

Lesions	Mycosis Fungoides	Hodgkin's Disease	Leukemia Cutis	Lymphosarcoma Cutis
Specific				
Papules	+			
Nodules	+	+	+	+
Plaques	+			
Tumors	+	+		+
Exfoliative erythroderma	+	+		
Nonspecific				
Any or all of primary skin lesions and/or				
Eczematoid lesions	+			
Psoriasiform lesions	+			
Zosteriform lesions		+	+	
Purpuric lesions			+	
Hemorrhagic lesions			+	
Increased pigmentation		+		
Exfoliative dermatitis	+	+	+	+
Pruritus	+	+		
Prurigo-like lesions	+	+	+	

In the terminal stage of each of the four forms of lymphoblastoma, the superficial and deep lymph nodes and the liver and spleen are often involved.

The specific skin lesions of these disease entities are true infiltrations into the skin of the cells characteristic of the disease. These lesions, papules, nodules, tumors, or plaques, are identified specifically only by microscopic examination; they are observed in any or all types of disease in the lymphoblastoma group and are constant neither in character nor appearance for any one disease entity.

The nonspecific lesions may consist of macules; papules; vesicles; bullae; and eczematoid, psoriasiform, herpetic, urticarial, purpuric, or hemorrhagic lesions. An increase in pigmentation, exfoliative dermatitis, and generalized pruritus are commonly found in all types of lymphoblastoma.

Cutaneous manifestations of lymphoblastoma and their accompanying visceral lesions as usually described are listed in table 3.

The cutaneous lesions and the four stages of mycosis fungoides, namely, dermatitis, infiltration, tumor formation, and ulceration, are characteristic. However, segments of this clinical pattern can be portrayed by any of the other lymphoblastomas. The dermal picture may appear to be classic mycosis fungoides at first but later prove to be Hodgkin's disease or lymphosarcoma. On the other hand, the initial cutaneous infiltrate which may seem to be that of Hodgkin's disease may terminate in a generalized lymphosarcoma involving the skin, lymph nodes, and viscera. The transformations which are possible among the four forms of lymphoblastoma are indicated by arrows in figure 1.

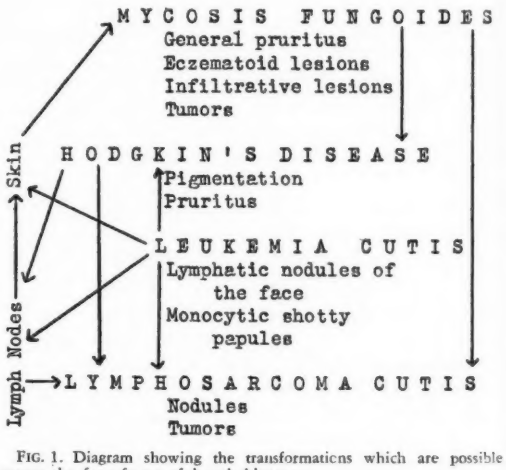

FIG. 1. Diagram showing the transformations which are possible among the four forms of lymphoblastoma.

The diverse cutaneous lesions manifested by the various forms of lymphoblastomas listed in table 3 are not clinically diagnostic but are suggestive. Only by correlating the dermal findings with pathologic, hemocytologic, and roentgenologic studies can the diagnosis be established. Biopsy is of great value in diagnosis of the various types of lymphoblastoma and, if not diagnostic, should be repeated as the disease changes.

There is no specific therapy for this group of diseases. In the light of present knowledge and experience, it can be concluded that chemotherapy with urethane and nitrogen mustard and radiation therapy, including irradiation of the whole body by the internal administration of radioactive isotopes of phosphorus, sodium, and magnesium, is merely palliative and not curative.

Roentgen irradiation is still the choice of treatment in all forms of lymphoblastomas and is most effective in mycosis fungoides. In many instances the patient with mycosis fungoides can be afforded great relief and his life prolonged many years by the judicious use of roentgen therapy.

In the treatment of Hodgkin's disease with cutaneous manifestation, roentgen irradiation is the method of choice, as involvement of the skin and glands usually is evidence of dissemination, which excludes surgical intervention.

In either mycosis fungoides or Hodgkin's disease, particularly the latter, the disease at times becomes roentgen-fast. Nitrogen mustard, methyl-bis (beta-chloroethyl) amine hydrochloride, may cause the recalcitrant disease to respond. After a short course of nitrogen mustard, the patient may again become sensitive to roentgen-rays and can be treated anew by the less toxic method.

Repeated courses of nitrogen mustard have fallen into disrepute because of the toxicity of the drug and often reactions from the treatment are worse than the disease.

Roentgen irradiation offers as much palliation as any of the newer chemotherapeutic adjuncts; it is certainly less toxic and causes less discomfort, particularly with Dramamine and Pyridoxine Hydrochloride, which can be used to relieve the radiation sickness.

DERMATOSES OF METABOLIC DISORDERS

Disturbances of protein, carbohydrate, and fat metabolism induced by various endogenous diseases are reflected as diverse entities in the skin.

Proteins—Nutritional Eczema

Guy, Jacob, and Guy[7] described a nutritional eczema occurring in the aged as an edema below the knees associated with varying degrees of acute or chronic inflammation of the skin, relative or complete achylia, and hypochromatic anemia. In all cases the total serum protein was diminished and the serum albumin-globulin ratio altered.

W. B. Guy[8] recently reported similar clinical patterns associated with liver diseases occurring in younger persons. There was a definite decrease of the total serum protein and an abnormal albumin-globulin ratio in these persons suffering from malmetabolism of proteins. Similar abnormalities in the composition of the blood were demonstrable in patients with indolent ulcers of the leg of hemostatic origin. If a patient has a chronic, edematous eczema or ulcers of the leg not explainable by the usual causes such as stasis or varicose veins, the protein metabolism should be investigated.

Guy and his associates reported that with the administration of protein hydrolysates, diets rich in protein, vitamins (yeast and liver fractions), and dilute hydrochloric acid and with the indicated local care, the clinical response in nutritional eczema was dramatic. In every instance, the total serum protein and the albumin-globulin ratio returned to normal.

Carbohydrates—Diabetes Mellitus

Cutaneous manifestations and symptom complexes referable to the skin which accompany uncontrolled and at times controlled diabetes are many and varied. These dermal signs and symptoms are not diagnostic but any one of them should alert the clinician to the possibility of this metabolic disturbance. They are as follows:

Diabetic Carotenemia.—Obvious generalized yellowing of the skin is one of the most common dermal reflections of this metabolic disturbance. The accompanying disturbed lipid metabolism and the inability of the patient to synthesize vitamin A from carotene, together with the faulty excretion of carotene, account for this peculiar discoloration of the skin.

Pruritus.—Generalized and localized itching are common cutaneous symptoms of hyperglycemia. When this troublesome complaint is localized to the perianal and genital region, it should especially arouse suspicion.

Pyogenic Infections.—Recalcitrant folliculitis, furunculosis, and carbuncles should always call to mind the possibility of a disturbance of glucose metabolism.

Fungus Infections.—Moniliasis, due to infection with the Candida albicans, frequently accompanies uncontrolled and controlled diabetes. Paronychia and yeast infections involving intertrigenous areas, as in the interdigital spaces, under the breast, axillas, groin, labial crevices and folds, and vaginal tract, are caused by this organism. This type of infection is noted frequently in uncontrolled diabetes and, at times, in controlled diabetes.

Xanthoma Diabeticorum.—The term is an incorrect designation because this yellowish papular or nodular lesion of the skin is an eruptive form of xanthoma and is due to the hyperlipemia associated with the uncontrolled diabetes; it has no direct relationship to the hyperglycemia. The characteristic small yellowish papules are usually grouped on the elbows and knees and over the buttocks. These lesions become involuted rapidly when the diabetes is controlled which, in turn, corrects the hyperlipemia.

Necrobiosis Lipoidica Diabeticorum.—From 80 to 90 per cent of the patients with necrobiosis lipoidica have diabetes; however, the dermatosis is also associated with other hyperlipemic states induced by hepatic and other visceral diseases such as nephritis. The characteristic lesion is a smooth, shiny, glassy,

yellowish-red, sharply demarcated plaque frequently coursed by fine telangiectatic vessels. In the majority of instances this entity occurs on the shins and around the ankles and is prevalent in women. The diagnosis is self-evident since in the majority of cases the lesion is so characteristic. If there be any doubt because of an unusual location or type of lesion, the clinical diagnosis can be substantiated by biopsy in 90 per cent of the instances,[5] as the histopathologic picture is usually typical.

In necrobiosis lipoidica the diabetes should be controlled; however, this alone does not seem to influence the condition. A low animal-fat diet in keeping with the strict diabetic regimen should be instituted. Lipotrophic substances are reputed to be of value in some cases. Vitamin E (tocopherol) in 200 to 300 mg. doses a day, as advocated by Burgess and Pritchard,[2] should be tried. I have noted some improvement in 2 patients with this form of therapy.

Diabetic Gangrene.—This complication occurs most frequently on the distal part of the extremities and on areas subject to trauma. It is more frequent on the lower extremities and at times occurs on the upper extremities, but rarely is any part of the body involved.

Diabetic Ulcer.—This painless, punched-out ulcer is similar in appearance and location to other trophic ulcers and usually develops at points of pressure.

Atrophy of Fat.—Atrophy of the subcutaneous fat occurs sometimes in diabetic patients and is found most frequently at the site of insulin injection; however, at times a loss of the subcutaneous fat also occurs in areas where the insulin was never given. These defects, usually multiple, are deforming and disfiguring depressions ranging in size from a 25 cent piece to a man's palm; they cause great mental anguish to the young diabetic patient.

The treatment is preventive. When loss of subcutaneous fat occurs at the site of injection, such loss is less likely to occur if a potent insulin, such as U 80, is injected deep with a long needle. In persons exhibiting this disposition, it is well that all injections be given in the wall of the lower part of the abdomen, where the deformity will not be seen so readily.

When loss of fat occurs in areas other than at the injection site, little or nothing can be done. These patients rarely have a low basal metabolic rate; in such an instance, however, thyroid therapy is reputed to be of some help in preventing this distressing dermal deformity.

The previously enumerated cutaneous complications and symptoms accompanying diabetes, coupled with an adequate history of the general symptomatology of this disorder and a general appraisal of the patient, merely suggest the possibility of a hyperglycemic state. The diagnosis rests on laboratory aid. The absence of sugar in the urine is inadequate evidence that the cutaneous problem in question has no relationship to diabetes. Even normal fasting blood sugar determinations and normal glucose tolerance curves do not completely dispel the possibility of cutaneous glycohistechia (isolated skin diabetes as described by Urbach[16]). At times the clinician has to resort to a therapeutic test such as low intake of carbohydrates and small doses of insulin to control some of the associated cutaneous problems and to establish that the dermal complaints are truly a cutaneous reflection of a disturbed sugar metabolism.

Fats—Xanthomas

The different kinds of xanthomas caused by a disturbance in the metabolism of fats are as follows:

Cutaneous Xanthoma.—A sorting of the cutaneous xanthoma according to usual anatomic situations is of great importance to the clinician, since the location often indicates the site of the associated visceral lesions; this relationship is exemplified by table 4 in which Thannhauser's classification[15] of xanthomatous disorders is followed.

Xanthoma of Eyelids.—Xanthelasma may be the only dermal xanthomatous lesion, or it may be associated with other plain or tuberous lesions in either the familial or secondary forms of xanthomas. For many years xanthelasma was considered a localized lipoidosis but Montgomery[11] helped disprove this belief by surveying 38 cases; he concluded that the condition is one of the types of xanthoma cutis and an accompaniment of the generalized xanthomatous process. Twenty per cent of the patients studied had serious cardiovascular diseases such as hypertension, coronary sclerosis, or angina pectoris.

Polano[13] also helped dispel the concept that xanthelasma was solely a localized fat disturbance by a survey which revealed that one-fourth of patients with this single dermal manifestation of lipoid disturbance had a high or otherwise abnormal amount of lipids in the blood. The investigation by Curtis and Berger[3] of a large series of patients with xanthelasma disclosed that 75 per cent of the patients had hypercholesteremia.

Xanthoma Disseminatum.—The millet seed-sized, reddish-yellow xanthoma of the eyelids accompanying similar and larger lesions on the flexural surfaces such as the neck, axillas, and bends of the elbows and knees constitutes the entity of xanthoma disseminatum. In this type of disease the xanthomatous infiltrates often invade the brain, especially near the pituitary gland, and frequently are associated with diabetes insipidus. There may be similar lesions in the bones, lymph system, mucous membrane of the pharynx and larynx, lung, pleura, liver, and spleen.

Xanthoma Tuberosum.—This xanthomatous process

commonly involves the cardiovascular system, especially the large vessels, aorta, endocardium, valves of the heart, coronary arteries, and large arteries of the legs, and results in grave cardiovascular disease. The importance of cutaneous xanthoma and the underlying concomitant systemic disease is forcefully emphasized by Montgomery's[10] statement that "... practically all patients with xanthoma tuberosum have hyperlipemia and hypercholesteremia and that in 40 per cent of the cases there is associated cardiovascular disease in the form of angina pectoris and coronary sclerosis or intermittent claudication and arteriosclerosis obliterans...."

Xanthoma in most of its clinical form shows a characteristic yellow or chamois color; therefore, little difficulty is encountered in diagnosis. Thannhauser brought out that xanthoma tuberosum could be distinguished from xanthoma disseminatum and secondary xanthoma of the eruptive nodular type by differences in location, color, and group arrangement; these differences are illustrated in table 4. In rare instances the lesion is maroon or reddish-brown; consequently the diagnosis possibly could be in question. In such instances a histopathologic study would aid.

Since 75 per cent of the patients with xanthelasma show a marked derangement of lipoid metabolism, reflected as lipemia and hypercholesteremia, a high percentage of these persons suffer or will develop cardiovascular disease. It is important that this malmetabolism of lipoids be recognized early and that corrective measures such as a rigid animal fat-free (low cholesterol) diet, be instituted. In many instances the blood will return to normal, and it is entirely possible that some of the early xanthomatous infiltrates in the vascular tree will regress. This involution can possibly be enhanced by lipotrophic medication.

The cutaneous lesions of xanthoma disseminatum show no response to any type of diet or medication but more important, the visceral xanthomatous processes are usually progressive until death.

Often in xanthoma tuberosum the cutaneous lesions become involute with the rigid animal fat-free (low cholesterol) diet and the blood chemistry returns to normal. It is possible that the early cardiovascular xanthomatous infiltrates are reversible on such a regimen or at least that further damage to the heart and vascular tree can be minimized and the patient's life prolonged. In addition to dietary measures, the administration of lipocaic or lipotrophic substances such as lecithin, choline, methionine may enhance the absorption of xanthomatous infiltrates in the large vessels. More active lipotrophic substances and true liposolvents may be developed which will afford more promise to these unfortunate persons suffering from malmetabolism of fats.

The secondary xanthomas become involute rapidly when measures such as control of the diabetes and relief of the obstruction of the common bile duct correct the accompanying hyperlipemia.

TABLE 4.—*Relationship of Cutaneous Xanthomas to Visceral Involvements.*

Location	Form	Lesion	Color	Arrangement	Visceral Involvement
Eyelids	Xanthelasma Xanthoma disseminatum	Plain papules	Chamois Lemon	Plaques, scattered	Cardiovascular No cardiovascular
Flexural surfaces Face Neck Axillas Cubital and popliteal fossae	Xanthoma disseminatum	Small shiny papules	Lemon to maroon	Clusters Ridges	No cardiovascular Brain Lymph system Bones Respiratory tract Liver and spleen
Extensor surfaces Elbows Knees Buttocks Back	Xanthoma tuberosum (familial)	Papules, plain or tuberous	Orange or carotene	Scattered, isolated, or grouped	Cardiovascular Heart Large vessels Tendons
Extensor surfaces, palms, and soles	Xanthoma secondary to liver disease Xanthomatous biliary cirrhosis Hemochromatosis Obstruction to common bile duct	Papules, plain or tuberous	Yellow		Bile capillaries Liver tissue
Extensor aspects Legs and arms Palms and soles	Secondary eruptive forms Uncontrolled diabetes Chronic pancreatitis Glycogen storage disease Lipid nephritis	Small papules with tiny umbilicated heads	Yellow, brown or reddish-brown	Always isolated	Cardiovascular tree and heart (if of long duration)

CONCLUSIONS

This hodge-podge array of cutaneous pictures and associated visceral lesions exemplifies the multiple ramifications and intricacies of some of the medical problems with predominating dermal manifestations.

This list of endogenous dermatoses could easily be increased by the hundreds.

The solution of these problems must be on an etiologic basis; therefore, the approach is medical. The dermatologist must seek the aid of the internist and laboratory in the diagnosis and solution of many dermatologic problems. Often because of his training and experience the dermatologist can lend a guiding hand in separating endogenous from exogenous dermal disease and can afford many short cuts in the diagnosis and solution of the medical problem.

On the other hand, many of these perplexing dermal entities may be solved only by the aid and cooperation of all medical practitioners and scientists.

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ABSTRACT OF DISCUSSION

DR. LESLIE M. SMITH, El Paso: Dr. Pipkin has put into his paper a great deal of information of which both the internist and the dermatologist should be constantly reminded. It would be impossible to cover all phases of such a subject in one paper of limited strength, and Dr. Pipkin has selected topics wisely.

It is now conceded that an increased androgen-estrogen ratio is necessary for the development of acne vulgaris. It is only in the presence of this relative excess of androgen that other factors such as faulty diet, anemia, and invasion of micro-organisms will produce the lesions. The comedo is a hyperkeratotic plug probably caused by androgenic stimulation. McKenna, Way, Andrews, Goeckerman, and others have reported on the benefits of estrogen therapy in acne vulgaris. In some cases, however, it has been necessary to give such large doses that the secondary sex characteristics in the male patients changed. Therefore, patients treated in this manner should be watched closely, and dosage should be conservative. With small doses of estrogen, untoward effects are not as likely. Further work along this line is necessary, and the guidance of an expert endocrinologist would be a great help to the dermatologist. The other factors in acne and the importance of appropriate local treatment should not be forgotten.

Psychosomatic diseases involving the skin are of growing importance and are actually on the increase. The feeling of stress and insecurity of the present has been a big factor in neurodermatitis and other psychosomatic disturbances in susceptible persons. Never was it more important for the physician to encourage vacations, hobbies, and regular diversions.

Patients with unexplained generalized dermatitis should be closely watched. Some of these, as Dr. Pipkin has intimated, are in the early stages of one of the forms of lymphoblastoma. In the dermatitis stage, biopsy will often, although not always, give a clue or at least alert the physician to the possibilities so that he will perform other biopsies. I have watched more than one case of apparently simple, even localized, dermatitis develop into serious lymphoblastoma. In these cases early biopsy, although not diagnostic, has usually called attention to the possibility of beginning lymphoblastoma.

The skin offers many warnings of internal disease, and often dermal symptoms may be the only warning. Many times they have been overlooked or disregarded. The value of a paper such as Dr. Pipkin's is that it makes physicians more alert and more receptive to these warning signs.

POSTGRADUATE SCHOOL OF MEDICINE

A third division of the University of Texas Postgraduate School of Medicine to be located in Temple was approved by the board of regents of the University on March 17.

According to an agreement with Scott and White Memorial Hospitals and Scott, Sherwood and Brindley Foundation, the Temple division will be located in and use the facilities of Scott and White Memorial Hospitals. Dr. G. V. Brindley, Sr., was appointed assistant dean for the Temple branch and members of the Scott and White medical staff were named members of the new faculty.

Emphasis will be given to the residency training program with twenty-six residents training in the medical, surgical, and radiological specialties. Refresher courses in general and special practice will be offered regularly.

The patient with tuberculosis must cure himself. The final conquest or destruction of the tubercle bacilli is a victory of the body itself. Physicians guide and assist the resisting forces of the diseased body against the rapid multiplication and spread of the invading germs. The general measures of rest and good nutrition remain basic in the treatment.—Calif. Med., John H. Skavlem, M. D., December, 1950.

INFERIOR OBLIQUE MUSCLE IN HEAD TILT

JOSEPH DUDGEON WALKER, M.D., Houston, Texas

HEAD tilt may be interpreted to mean any condition wherein the head no longer assumes the vertical position. True ocular head tilt must have as its cause a heterophoria or heterotropia with or without manifest diplopia, and the cause is nearly always due to paresis of an elevator or depressor muscle.

Photography has done much to illustrate to the busy ophthalmologist head tilt due to vertical muscular imbalances which otherwise might have gone unobserved. In my early introduction to muscle surgery of the eye, I recall a photographer pointing out that people who came to him for pictures did not always tilt their heads to the side merely to be coy, but to cover up facial defects or to bring the eyes level. In the latter instance, the photographer perhaps unknowingly was thinking in terms of ocular torticollis, due to a paresis of one or more of the oculorotary muscles.

Yater's textbook of symptom diagnosis[21] lists numerous causes for head tilt such as cerebral tubercular meningitis, retropharyngeal abscess, cervical fibrositis, and semicircular canal lesions, without any mention of ocular torticollis because of nystagmus, wherein the head is turned so as to put the eyes in the field of the slower component; uncorrected oblique astigmatism of high degree; a bilateral ptosis producing a backward tilting of the head with chin elevated; or a paresis (weakness) of an elevator or depressor muscle.

In preparation for surgery to correct head tilt due to hyperphoria, I first do a careful refraction, red Maddox rod study, diplopia field studies with red glass, parallax tests, cover test, version studies, and measurements of esophoria, exophoria, and hyperphoria with square glass prisms in the primary position at 6 M. and at 33 cm. in the six cardinal fields. A square glass prism is used because it can better be balanced so the patient looks exactly through its center; hyperphoria as measured with the square prisms frequently shows greater deviation than the diplopia field would indicate. A differential diagnosis of paresis of elevators and depressors is made in an effort to determine (1) the paretic muscle, (2) the overactive muscles, and (3) the muscles with inhibitional palsies and the muscles that have developed secondary contractures.

White[15] set forth indications for surgery upon the inferior oblique muscle, which to date have not been changed materially:

Indications.—(1) paresis of the superior rectus muscle of the contralateral eye; (2) paresis of the superior oblique muscle in which the inferior oblique muscle has developed a secondary contracture; (3) primary spasm of the inferior oblique muscle; (4) paresis of the superior rectus muscle with 5 prism diopters or more of hyperphoria in the primary position with an increase to 10 or more in the field of the paretic muscle, surgery being confined to the field of the paretic muscle.

Contraindications.—(1) one superior rectus muscle almost completely paralyzed (surgery should be confined to the paretic superior rectus); (2) both superior recti muscles almost completely paralyzed (no surgery of the inferior oblique recti muscles should be done because they must be preserved for elevation; resection or advancement of the superior recti muscles is indicated).

SURGICAL CORRECTION

Before operating on the inferior oblique muscle for correction of hyperphoria, I prefer to correct surgically any esophoria that exists. However, exophoria, unless extreme, is corrected at the time the tendon of the inferior oblique muscle is recessed. I have observed hyperphoria to disappear after the horizontal muscle balance is corrected. This reaction occurred in one case following the Jameson type recession of the internal rectus muscles for convergent strabismus. A skew deviation of the overacting right internal rectus muscle existed in the presence of 45 to 60 or more degrees of adduction. The contraction of the internal rectus muscle produced maximum intorsion and adduction, resulting in the inferior oblique muscle becoming overstimulated and "spastic" and thus causing extreme hyperphoria. This hyperphoria disappeared after recession of the internal rectus muscle (fig. 1).

In every instance I first operate upon the weaker or habitually deviating eye, which is usually the nonfixing eye. In the presence of an overacting or spastic inferior oblique muscle I choose to correct the hyperphoria by a recession or partial tenotomy of the inferior oblique muscle. In cases in which recession or tenotomy is not sufficient to correct the hypertropia, I either resect the superior rectus or recess the inferior rectus muscle of the contralateral eye.

In a paresis of the left superior oblique muscle, the head is tilted to the right with chin twisted to the left, because when the head is tilted toward the right shoulder a parallel rotation of the eyes is produced around the visual axis to the opposite side, that is,

Read before the Section on Eye, Ear, Nose, and Throat, State Medical Association of Texas, Annual Session, San Antonio, May 4, 1949.

the vertical axis remains vertical so long as the head is tilted to the right. When the patient is forced to turn his head toward the paralyzed left superior oblique muscle, the intorsional effect of the superior oblique muscle is lost, leaving the intorsion, elevation, and adduction of the superior rectus muscle unopposed; thus the left eye rotates upward. This reaction also is evident when the patient maintains his head in the vertical meridian. The paralyzed eye remains slightly deviated upward, except when the head is sufficiently turned to the right to permit rotation of the eyes around the visual axis. The deviation is as marked when the patient is looking up and to the right as it is when he is looking down and to the right. For this reason a recession not to exceed 10 mm. of the inferior oblique muscle of the left eye should be done simultaneously with a recession of the inferior rectus muscle of the right eye. If this patient had only a paresis of the superior oblique muscle of the left eye, with the deviation definitely more marked in the field of the paretic muscle, the inferior rectus muscle of the opposite eye might be recessed without tenotomy or recession of the tendon of the inferior oblique muscle, at least for the moment.

FIG. 1. Results obtained in a case of esophoria complicated by hyperphoria treated surgically by a bilateral, Jameson type recession of the internal rectus muscle. Above are the patient's eyes before surgery; center, ten days after surgery; and below, seventeen years after surgery.

With the head in the primary position, the deviation in paresis of the left superior oblique muscle increases when the patient looks down and to the right, because the principal limitation of movement is when eyes are down and to the right. The diplopia is vertical and torsional, the vertical separation of the images increasing upon looking down and to the right.

My basis for treatment of head tilt in a case of paresis of a left superior oblique muscle is as follows: If the left hyperphoria or hypertropia is 5 prism diopters or less, a prism correction of three-fourths of the amount of prism deviation is prescribed, base down in front of the left eye and base up in front of the right eye, or one-half of the prism power divided between the two eyes, this being three-fourths of the total diopter deviation; however, this procedure helps only in the central field. If the left hyperphoria measures from 5 to 10 prism diopters, a recession of from 3 to 5 mm. of the opposite inferior oblique muscle of the same eye is recommended. However, if the left hyperphoria is extreme, a guarded tenotomy may be done. I never do a complete tenotomy upon the inferior oblique muscle or upon any extraocular muscle. Sometimes I free the check ligaments of the inferior oblique muscle where they are continuous with the check ligaments of the inferior rectus muscle and where fascial strands indirectly find their way to the orbital ridge; at the same time I recess the inferior rectus muscle of the right eye.

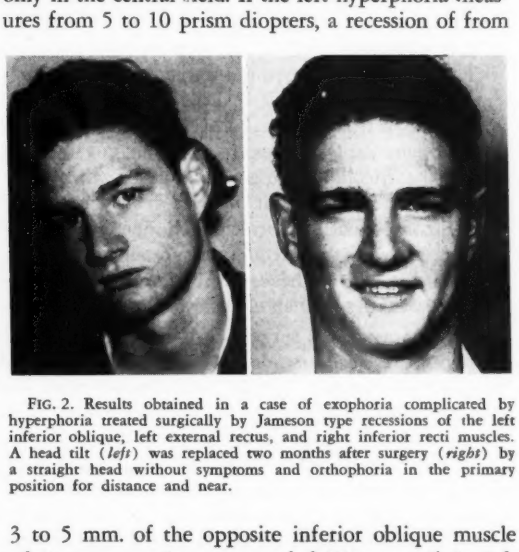

FIG. 2. Results obtained in a case of exophoria complicated by hyperphoria treated surgically by Jameson type recessions of the left inferior oblique, left external rectus, and right inferior recti muscles. A head tilt (*left*) was replaced two months after surgery (*right*) by a straight head without symptoms and orthophoria in the primary position for distance and near.

One patient showed a left hyperphoria of 25 prism diopters with eyes down and right, a left hyperphoria of 28 prism diopters with eyes up and right, and an exophoria of about 20 prism diopters in all fields.

A 7 mm. recession of the left external rectus muscle with an 8 mm. recession of the left inferior oblique muscle was performed, together with a recession of 3.5 mm. of the tendon of the right inferior rectus muscle. The results were successful (fig. 2).

The whole story regarding the overall muscle picture is not evident until the muscles are exposed at operation. Occasionally the check ligaments are found definitely interfering with the normal action of the muscle, especially those of the external and internal recti muscles. The inferior oblique muscle also may have its share of check ligaments.

SUMMARY AND CONCLUSIONS

What can the ophthalmologist promise a head tilt patient with hyperphoria or hypertropia after surgery upon the inferior oblique muscle? Head tilt can be corrected by proper surgery upon the inferior oblique muscle if it is caused by hyperphoria or hypertropia.

Liberal surgery may be performed if the patient is known to have fusion, provided a correct interpretation of the action of the offending muscle or muscles is made. The offender may be the paretic, the antagonist, or the yoke muscle, depending upon which is the dominant or fixing eye.

Ophthalmologists must be cognizant of the fact that patients sometimes spontaneously regain normal retinal correspondence with normal binocular vision, or normal sensorial orientation with attending normal and anomalous localization without the development of binocular vision. However, the majority of patients stabilize themselves in an anomalous sensorial relationship adapted to the motor condition. Even after surgery patients may retain, at least in the after image test, the anomalous correspondence acquired previous to operation.

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ABSTRACT OF DISCUSSION

DR. E. MEREDITH SYKES, San Antonio: Estimation of the action and function of the extraocular muscles has always been a challenge to the ophthalmologist who would align visual axes by any means—operative or nonoperative.

The action of any single muscle or group of ocular muscles is dependent on the individual size, innervation, and site of the tendinous attachment and check ligaments, as opposed by similar or dissimilar qualities in the antagonists. Atrophies, hypertrophies, pareses, and contractions or certain muscles generally place any surgical procedure for the correction of tropias or phorias in the realm of approximation rather than of mathematical precision. Surgical judgment—a rare quality —comes from long experience with ocular muscle cases.

A few years ago as a spectator in the operating room of a well-known New York hospital I heard the lamentations of a young but skillful ophthalmologist who had operated in a case of esotropia. He had done a recession of the internus and a resection of the externus muscle on the same eye, but the result was an eye turned outward. An older and more experienced surgeon made the wise criticism that operating "according to Hoyle" would sometimes end badly.

The surgical correction of the essayist's case of esophoria complicated with hyperphoria and torsion demonstrates the pertinent fact that the change of position of the eyeball following a recession of the internus muscle also changes the leverage and action of other muscles and also can relieve a muscular spasm, as in his case, of the inferior oblique muscle, which was the causative factor producing a distressing head tilt. It is generally conceded that a recession of the internus muscle has a more profound effect than the setting back of any of the other ocular muscles.

Occasionally, head tilting is seen in patients who have a habit of cocking the head to one side or the other without having any ocular muscle disturbance as a causative factor. This has been noted in children imitating a parent who has developed this type of head tilting. However, as a general rule head tilting is associated with a vertical and rotary imbalance, one of the oblique and/or the superior or inferior recti muscles being the muscles usually at fault.

It has been pointed out by Bielschowsky and Adler that the direction of the tilt will always be toward the opposite shoulder when the superior rectus or oblique muscle is involved; whereas, when the inferior oblique or inferior rectus muscle is involved, the direction will be toward the same shoulder. Bielschowsky's sign is a further diagnostic point when a superior oblique muscle is paretic.

NASAL ALLERGY

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FOR many years otolaryngologists have been coping with nasal obstruction and drainage. Many surgical procedures have been used and many drugs applied to the nasal mucous membrane in an effort to eliminate symptoms but in spite of such concentrated attacks, obstruction and drainage have remained the most common nasal complaints. A glance at the failures of the past should convince even the most skeptical that infection is not the major etiologic factor. More than a decade ago Hansel[1] pointed out that allergies were commonly the cause of nasal disorders. Since then many outstanding investigators have verified his claim and have directed the trend of nasal therapy along more rational lines.

DIAGNOSIS

The diagnosis of nasal allergy can be made after a careful history, a thorough examination of the nose, a study of the nasal secretions, and skin tests. Of these, the history is the most important.

In my opinion, nasal obstruction is the most common complaint. It may be constant or intermittent and in many cases changes from side to side. Excessive nasal drainage is the second most common complaint. The discharge may be watery or thick and tenacious; it is clear most of the time but occasionally becomes yellow. Sneezing may or may not be present. An itching sensation in the posterior nasal passages may be noticed. Headache, fullness in the ears, irritation of the throat, and cough are often primary or complicating complaints.

In the history the physician should accurately determine if there is a seasonal incidence or a night and day variation in the symptoms. Home and occupational contacts with dust, chemicals, animals and smokes should be ascertained. Symptoms which are associated with the ingestion of certain foods should be inquired about. A review of dietary habits will reveal foods that are eaten in excess and those that produce minor gastrointestinal upsets. It is significant to note whether or not the symptoms remain constant upon a complete change of environment. A family history of any type of allergy is important. The patients should be questioned as to drugs being taken and known drug sensitivities. Nasal drops and sprays are so commonly used today that their type and frequency of application should be determined. The nose is an integral part of the body, not an isolated structure, and no history may be considered complete until a few questions are asked to evaluate the patient's general health. Such questions should determine the menstrual history, the presence or absence of excessive fatigue, excessive nervousness, constipation, major operations, major injuries, alcoholism, insomnia, and emotional status.

In the uncomplicated case the rhinoscopic examination usually reveals little pathologic change. At one time a pale, boggy mucous membrane was thought to be diagnostic of allergy and a hyperemic mucous membrane was thought to indicate infection. Improved methods of diagnosis have demonstrated that these assumptions are not necessarily correct. The allergic membrane may be pale and boggy, normal in appearance, or hyperemic. The nasal secretion may be scanty or abundant or thin or mucoid in consistency and may vary from clear to cloudy to yellow in color. The presence of polypoid tissue is an important diagnostic finding. Deviations of the nasal septum and the degree of obstruction produced should be determined. True hyperplasia of the turbinates should be differentiated from edematous turbinates which shrink well with vasoconstrictors.

A cytologic study of the nasal secretion must be made before an examination is considered complete. The secretion may be collected by having the patient blow his nose on a piece of waxed paper. Nasopharyngeal secretions may be obtained by using a suction tip through the nose or by elevating the soft palate and grasping small pieces of the secretions with the bayonet forceps. The secretions are placed on a glass microscopic slide, fixed by heat, and stained with Hansel's[4] quick stain; microscopic examination of the stained preparation should be made by the examiner. It takes considerable experience to evaluate properly the significance of the cell ratio between eosinophils and neutrophils, and this evaluation must be coupled with the history and physical examination of the patient. When the physician relies upon the report of a laboratory technician, the value of this all-important correlation is decreased.

The stained preparation from a normal nose will show epithelial cells, a few neutrophils, and an occasional eosinophil. In cases of uncomplicated allergy the preparation will show large numbers of eosinophils, epithelial cells, and an occasional neutrophil. The microscopic study in infection will reveal many neutrophils, epithelial cells and an occasional eosinophil. Allergy with secondary infection will cause many neutrophils, an increased number of eosinophils, and a few epithelial cells to be noted in the preparation. In many instances it is necessary to repeat nasal

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smears in order to establish the correct diagnosis. This is true because of the fact that the neutrophil response in infection is so much greater than the eosinophil response in allergy. As the infection subsides, successive smears will show the basic eosinophilia.

Other conditions such as the excessive use of vasoconstrictors in the nose, endocrine imbalances, and emotional upsets simulate nasal allergy. It must be borne in mind that one or more of these conditions may exist in conjunction with a nasal allergy.

Nasal sprays and drops nearly always produce vasoconstriction that lasts from two to four hours. After this initial action, a secondary vasodilatation always occurs. When this cycle has been completed several times per day for many weeks, the patient complains of severe nasal obstruction and drainage. The physical findings are usually the same as those in a true nasal allergy and the nasal smear is normal. The history will reveal the type of drug and the frequency and duration of its use. It is also important to determine why the patient began to use the vasoconstrictor.

Wright[7] is of the opinion that the synthetic preparations are more likely to produce a severe secondary vasodilatation than are the natural products. I have noted Privine and 1 per cent Neosynephrine to be the most common offenders. Withdrawal of the offending drug will eliminate this condition.

Proetz[6] has observed an apparent correlation between the thyroid gland and the nose. A deficiency in the hormone from this gland will intensify the symptoms of nasal allergy. Therapy for allergy will fall short of its desired goal unless this complicating condition is recognized and controlled.

Again, the history gives the first clue that a thyroid deficiency may be present. One or more of the following symptoms is usually present: excessive fatigue, intolerance to cold, increased desire to sleep, dry skin, and brittle nails. The physical findings are the same as in cases of uncomplicated nasal allergy. Eosinophils will be present in the nasal smear in direct proportion to the degree of allergy.

The basal metabolic rate has proved so inaccurate that I prefer to rely upon the clinic response obtained by giving small amounts of thyroid extract. In adults .25 grains, the usual initial dose, is increased by .25 grains every two weeks until clinical improvement is noted. Rarely has a total dosage in excess of 1.0 grains per day been necessary.

The importance of psychosomatic medicine is being more clearly recognized each day. Wolff[5] has been able to produce nasal obstruction and drainage by causing patients to be faced with difficult life situations. In a few patients he has charted the course of nasal disease in relation to emotional upsets. The nasal changes may be identical with those of a true nasal allergy and the two conditions must be differentiated, yet it must be remembered that both may be present in the same patient at the same time. Psychosomatic disorders of the nose can best be determined by the history and the nasal smear. Psychiatry is a complex specialty, and although otolaryngologists are not expected to be experts in the field, they should be alert to recognize emotional disturbances and place such patients in the hands of competent psychiatrists.

Skin Tests

Skin testing to determine the specific antigens causing the allergic symptoms is still of great value. These tests have fallen into partial disrepute because the results are often inadequately interpreted for the patient and because of a tendency to test for too many substances. Such tests serve well as a guide but may not be used as an exact basis for diagnosis and treatment.

The most common methods are the scratch, intradermal, and punch tests. Punch tests are well tolerated by children but are not as accurate as the other methods. Many allergists use the scratch tests for pollens and as a preliminary test for inhalants and certain foods such as oils, fats, nuts, fish, and albumins. It is generally conceded that the intradermal test is the most accurate, and I believe that it can be safely performed by experienced personnel without preliminary testing if adequate dilutions are used.

The following safety rules should be observed:

1. Asthmatic patients should be tested with solutions which are weaker than normal.
2. Sensitivities revealed by the history should be tested for with weak solutions.
3. All tests should be applied to the arms and in the event that systemic reactions occur, a tourniquet should be placed above the test sites.
4. The number of tests made at any one time should be limited by the severity of the reactions.
5. Adrenalin should be readily available for emergencies.

Whether the patient's complaints be seasonal or perennial, tests for the inhalants, foods, pollens, and in some cases for bacteria should be made. About 70 per cent of patients with pollen sensitivity also will have inhalant or food reactions. The inhalant and food sensitivities may not be strong enough to produce symptoms but coupled with the pollen factors will sometimes cause the pollen treatment to fail. When patients who are supposedly allergic only to pollen do not become symptom-free after a heavy frost, food and inhalant sensitivities should be suspected.

To avoid unnecessary testing, field trips to determine the most prevalent trees, grasses, and weeds in a given geographic area should be made. Daily pollen

counts will show the time and degree of pollination. The majority of perennial nasal allergies are caused by the incidental group of inhalants; the most common offenders are house dust and molds. House dust is considered to be a combination of molds, fabric fibers, tobacco, animal hairs, and fungi. Therefore, testing for incidentals must necessarily be the most comprehensive of all. The patient's history and dietary habits should guide the selection of foods for testing.

TREATMENT

Pollen sensitivities may be treated by the preseasonal or coseasonal methods. If the patient reports for treatment prior to the onset of the pollen season, I prefer the preseasonal method, which consists of a series of injections of the offending allergens, starting with small doses and gradually building to higher levels. The degree of skin sensitivity and the severity of the symptoms are guides for establishing the initial dose. Usually .1 cc. of a 1 to 10,000,000 dilution is a safe initial dose. The vaccine is increased by .1 cc. each dose and an injection given every other day. Most patients are controlled at the 1 to 1,000 dilution level, after which injections may be given at weekly or biweekly intervals with no further increase in dosage. To prevent local and systemic reactions patients treated by this method must be carefully observed.

The coseasonal method of treatment is used when the patient reports during the pollen season. This method is based on Hansel's[2] determination that a certain optimal dose will cause the patient to be symptom-free for from one to three days. Intradermal titrations are used as a guide in determining the optimal dose and then the vaccine is administered as often as needed. The weaker concentrations used in this method minimize dangerous reactions.

In perennial nasal allergy, hyposensitization is the treatment of choice but offending allergens should be eliminated as far as possible. Patients should be instructed to cover pillows and mattresses with allergy-proof covers. The bedroom should be stripped of rugs, drapes, woolen blankets, and other objects that accumulate dust. Cotton blankets should be washed at least one time per week. The floors and woodwork should be wiped with an oily cloth at frequent intervals. Nonallergic cosmetics should be used. Animals, insect sprays, chemical fumes, strong soaps, and smoke should be avoided by the patient.

When elimination alone does not relieve symptoms, dust vaccine is indicated. Various molds, animal danders, smoke, and other inhalants may be added to the dust vaccine as indicated by the history and skin tests in the ratio 9 parts dust to 1 part inhalant. The low dosage method used by Hansel[3] has been the most satisfactory in my hands. The initial dose is based on the patient's symptoms and not on the degree of skin sensitivity; the more severe the symptoms, the smaller the initial dose. A good starting point in severe cases is .1 cc. of a 1 to 1,000,000,000 solution. Injections are given at four-day intervals and increased .05 or .1 cc. each time until the patient becomes symptom-free. At this point the strength of the dose is not changed and the interval between doses is increased to one week, then two weeks, then three weeks, the goal being to determine the optimal dose that will maintain a given patient symptom-free for three weeks. After three three-week periods, the patient is discharged with the instruction to return if symptoms recur.

In recent years the antihistaminic drugs have been widely used in allergic conditions. These drugs are helpful in pollen allergies because the histamine factor is prominent but are of little value in the ordinary low-grade case of perennial allergy in which histamine is a minor factor.

Food allergies can be successfully treated only by removing all offending foods from the diet. Because of the inaccuracy of skin tests for foods, the history plus the use of elimination diets offers the best means of determining these offenders.

SUMMARY

Allergy is a common cause of nasal obstruction and drainage.

A complete history is all-important as a diagnostic aid. No examination is complete until a cytologic study of the nasal secretion has been made.

Nasal symptoms produced by chemical irritations resulting from the excessive use of vasoconstrictors, by endocrine imbalances, and by emotional upsets must be differentiated from true allergies.

Skin tests are important to indicate sensitivity to specific allergens, but they are not infallible and therefore should be used only as a guide. Pollen sensitivities may be treated by the preseasonal or coseasonal methods.

The inhalant group is the most common cause of perennial nasal allergy, and house dust is the most common offender in this group. In its treatment elimination should be coupled with hyposensitization by the low dosage method.

Food allergies must be treated by elimination.

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ABSTRACT OF DISCUSSION

DR. JAMES P. GILL, San Antonio: Dr. Hall has completely covered the main points regarding the symptoms, diagnosis, and treatment of nasal allergy.

Sulfonamides and antibiotic drugs do not influence the allergic group of patients. The antihistamines are of value in some cases.

In acute cases I use an intravenous injection of calcium gluconate, 10 per cent solution, which helps temporarily to alleviate symptoms. This treatment is supplemented by subcutaneous injections of parathyroid extract, .5 cc. twice weekly.

In certain selected patients in whom symptoms are quiescent, the correction of marked septal deformities and the relief of obstruction from nasal polyps are essential.

PRESSURE TREATMENT OF PURULENT MAXILLARY SINUSITIS

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THE treatment of maxillary sinusitis by the application of local pressure to the mucosa of the sinus was developed primarily to promote the absorption of fluid from edema in the interstitial spaces of the sinal mucosa during allergic sinusitis. Removal of this fluid has greatly reduced the systemic absorption and postnasal drainage of substances which are factors in intensifying the bronchopulmonary complications of allergic sinusitis such as cough, bronchitis, and bronchial asthma. During the use of this procedure for allergic sinusitis and allergic sinusitis with secondary infection, it became apparent that the signs and symptoms of the secondary infection were the first to disappear as the disease responded favorably to treatment. After this observation, patients with primary subacute and chronic purulent maxillary sinusitis were treated by the application of local pressure to the mucosa of the maxillary sinuses.

The results of this type of treatment in properly selected cases have been comparable and in certain instances superior to those obtained in similar cases by the radical maxillary sinal or Caldwell-Luc operation. The better results obtained by the application of pressure to the sinal mucosa apparently have been due to the more nearly complete cure of the sinal infection, which is evidenced by the absence of purulent secretion from the sinus, the return of all or most of the function of the ciliated sinal epithelium, and the restoration of normal transillumination of the sinus. Although the radical maxillary sinal operation is probably the most uniformly successful sinal operation performed by the rhinologist and therefore is definitely indicated for certain types of sinal disease, some patients present anatomic or pathologic conditions such as sinal septums or developmental irregularities which preclude a complete cure by the operation. The difference between complete and incomplete cure of maxillary sinusitis is particularly important in those patients who have or who are beginning to develop bronchiectasis as a consequence of the postnasal drainage of purulent sinal secretion.

The criteria used to select patients suitable for pressure treatment are based on clinical observations and the physiologic, functional pathologic, and histopathologic condition of the sinal mucosa. Usually the chief criterion for advising the radical operation is the persistent formation of purulent secretion in the sinus after all the more conservative therapeutic measures have been used. Proper interpretation of observations on the type and quantity of secretion evacuated from the maxillary sinus during irrigation is of prime importance in judging the character and extent of pathologic changes in the sinal mucosa.

During acute purulent sinusitis not caused by osteitis, osteomyelitis, foreign bodies, or dental caries, the secretion usually passes through serous, purulent, and mucoid stages as the infection is resolved. If the orderly progress of these changes in the secretion is delayed or if the mucoid stage does not occur, usually the infection has produced toxic or structural changes in the sinal mucosa which have interfered with the function of the cilia or the nutrition of the mucous glands. These changes also may interfere with the structure and function of the sinal mucosa by limiting the ability of the sinal mucosa to become edematous and approximate the capacity of the sinus, thereby forcing the secretion to drain through the sinal ostium.[2] As the sinal mucosa becomes fibrotic and loses its ability to become edematous in response to infection and as the nutrition of the mucous glands and ciliated epithelium becomes impaired, the sinus tends to become a septic cavity harboring stagnant decomposing secretion which often is secondarily infected by saprophytic organisms. Drainage of this type of secretion into the trachea and bronchi frequently is the cause of pulmonary disease, which

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usually begins with persistent cough and years later eventuates in the progressive pathologic changes of chronic bronchitis, bronchiectasis, and pulmonary suppuration.

As purulent maxillary sinusitis passes through acute, subacute, and chronic stages, biopsy of the sinal mucosa shows three types of pathologic changes. During acute purulent maxillary sinusitis, the interstitial spaces of the sinal mucosa are widely dilated with edema fluid, and the lymph channels show an accumulation of fibrin, cellular detritus, and round cells. As the sinusitis passes into the subacute stage, much of the fluid, fibrin, and cellular detritus is absorbed, leaving profuse round cell infiltration of the interstitial spaces and hyperplasia of the epithelium as the dominant pathologic changes.

FIG. 1. Radiograph showing balloons inflated with iodized oil in the maxillary sinuses. Distention of the balloons is accomplished gradually as their pressure against the edematous sinal mucosa promotes absorption of the excess extravascular fluid.

The pathologic picture observed during the subacute stage may last for years before the nutrition of the mucosa becomes affected. Clinically, the subacute stage of maxillary sinusitis may be classified erroneously as the chronic stage because of a long period of infection. However, it is not unusual for a patient to give a history of maxillary sinusitis of from ten to fifteen years' duration, only to observe upon biopsy of the sinal mucosa that the sinusitis is still in the subacute stage and that the pathologic changes are completely reversible. The best results from the application of local pressure to the mucosa of the maxillary sinus are obtained during the subacute stage of infection while the pathologic changes are still reversible.

The most important pathologic change during the chronic stage is fibrosis of the stroma of the mucosa which, as it becomes extensive, interferes with the nutrition of the mucous and serous glands and the ciliated epithelium. As fibrosis occurs, the stroma becomes denuded of epithelium and the mucosa suffers irreparable damage; this process usually surrounds a septic cavity.

Biopsy of the sinal mucosa is not always feasible. Therefore, it is often necessary to rely entirely upon clinical examination, which always should include observations of the type, quantity, and rapidity of formation of the sinal secretion obtained by repeated sinal irrigation. A careful inquiry into the patient's history, including episodes of pulmonary disease occurring during infancy and childhood, and roentgen studies of the sinuses and chest with and without contrast media are indispensable parts of the clinical examination. Bronchopulmonary disease in early life may predispose to the development of bronchiectasis during prolonged purulent sinusitis in the adult.

TYPES OF PATIENTS

Clinical examination reveals that most patients with purulent maxillary sinusitis may be divided into one of five groups. These groups are not offered as a classification but rather as a practical clinical aid useful in deciding the suitability of certain patients for the application of local pressure to the mucosa of the maxillary sinuses. The groups are as follows:

1. *Acute purulent sinusitis.*—Any acute infection in the sinuses, whether it be initial or an acute exacerbation of a previous infection, is a contraindication to the application of pressure to the sinal mucosa. If the mucoid stage of the sinal secretion does not appear or if it persists after primary acute purulent sinusitis, often the delay in resolution of the infection is the result of the treatment or lack of treatment. Ineffective treatment usually is due to one of three factors:

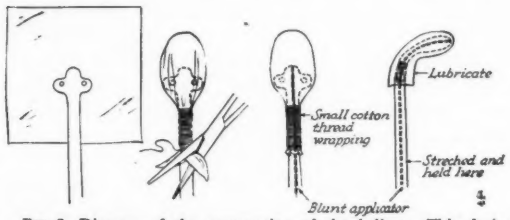

FIG. 2. Diagram of the construction of the balloon. This device essentially consists of enclosing a deformable self-retaining catheter head inside a distensible rubber balloon. The diagram to the far right shows the catheter head deformed over a curved metal obturator and ready for insertion through an antrotomy under the inferior turbinated bone. The size of the self-retaining catheter head may be varied to conform to the size of the intranasal antrotomy. This appliance may be made according to the diagram or obtained commercially (C. R. Bard, Inc., Summit, N. J.).

a. Reinfection of the maxillary sinus by bacteria induced during irrigation. Nothing which cannot be injected subcutaneously should be introduced into the maxillary sinus. In acute purulent maxillary sinusitis, if the maxillary sinus is irrigated after puncture under

the inferior turbinated bone, usually the injected fluid enters first into the edematous sinal mucosa. If the solution is contaminated or irritating, it hinders rather than promotes resolution of the infection. To avoid this possibility, it is best to irrigate only with normal saline solution which has been sterilized in an autoclave.

b. Injection of irritating solutions. Intrasinal medication by injection may damage permanently the sinal mucosa. This damage is seen most often after injection of a suspension of one of the sulfonamides; in this form the drug may be left in a localized area of sinal mucosa which may become surrounded by gangrenous mucous membrane. Penicillin injected into the sinus or sinal mucosa may continue the inflammatory reaction as a result of an allergic contact mucositis, or in strong solutions it may paralyze the cilia of the sinal mucosa. This paralysis prolongs the emptying time of the sinus and leads to persistent retention of secretion, which at first may be sterile but later may become infected. An aqueous solution of sodium penicillin G should never be injected into the maxillary sinus in a concentration stronger than 1,000 units per cubic centimeter; some evidence is accumulating that even this strength may be harmful to the sinal cilia. In certain patients in whom resolution of the sinusitis is delayed nothing but normal sterile saline solutions should be injected.

c. Irrigation of the maxillary sinus through the natural ostium instead of puncture under the inferior turbinated bone. Many patients with acute purulent maxillary sinusitis can be cured promptly and completely by irrigation through the natural ostium of the sinus. Contrariwise, some types of secretion in certain stages of sinusitis cannot be removed through the natural ostium and can be evacuated only by puncture and irrigation under the inferior turbinated bone.

2. *Subacute purulent sinusitis.*—Most patients who comprise this group give a history of having had several acute attacks of sinus infection from which they apparently recovered, then one attack which persisted. The most frequent error made in the diagnosis of subacute purulent sinusitis is to place emphasis on the duration of the sinal disease. Numerous factors determine the rapidity with which irreversible structural changes in the sinal mucosa develop. Although the duration of the infection in certain instances may be important, it is the most misleading factor in the clinical picture. This conclusion is borne out by the clinical observation that some patients have had purulent secretion in the maxillary sinuses for years with complete absence of symptoms and have recovered completely without treatment. Also, certain other patients who have had purulent secretion in the maxillary sinuses for a long period recover completely after a few irrigations or, if recovery is not complete, the character of secretion changes to the mucoid type.

The most important clinical factors which are useful in diagnosis of the subacute stage of infection are the ability of the sinal mucosa to form mucoid secretion, remove extraneous substances from the sinus by ciliary action, and become edematous.[3] Roentgenologic examination using a contrast medium before and after treatment often reveals the ability of the sinal mucosa to become edematous and increase its volume until it approximates the capacity of the sinus. Also, injection of irritating solutions into the sinal cavity may increase or produce edema of the mucosa.[7] Repeated injection of concentrated solutions of penicillin is contraindicated as a therapeutic measure. As a diagnostic

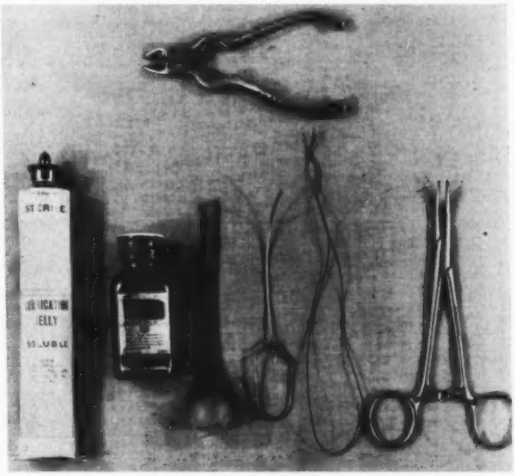

FIG. 3. Accessories used to insert a balloon into the maxillary sinus. From left to right are shown the lubricant used on the balloon and obturator, the iodized oil used to inflate the balloon, the balloon, the obturator used for deforming the catheter head and inserting the balloon, the string which ties the tip of the catheter, and the hemostat used to grasp the catheter after inflation of the balloon; above is seen the manicurist's clippers used to clip the string and catheter. The sharp-pointed clippers are useful to cut the string around the catheter when it becomes necessary to add oil to the balloon.

procedure, however, roentgen examination before and after one injection of 5 cc. of sodium penicillin G, 25,000 units per cubic centimeter, has been useful in determining the ability of the sinal mucosa to become edematous.

3. *Chronic purulent sinusitis.*—The clinical diagnosis of this stage of the disease should be made only after all conservative therapeutic measures in the sinus fail to stop the formation and retention of purulent secretion. Also, before the diagnosis is confirmed, one or more biopsy specimens should be studied with particular reference to the condition of the ciliated epithelium and mucous glands and the degree of fibrosis of the mucosal stroma. In preserving certain specimens, it is important to place the tissue between pieces of liver to protect the epithelium from injury

by being sloshed and traumatized in the preservative solution.

4. *Sinusitis with fetid secretion.*—This type of secretion may be present during acute, subacute, or chronic purulent maxillary sinusitis. Often when fetid homogenous purulent secretion is evacuated during irrigation of the sinus, the first impulse is to classify the case as one of severe infection which has probably produced irreversible structural changes in the sinal mucosa. This assumption is frequently erroneous and should not be acted upon until all conservative therapeutic measures fail to resolve the infection. Fetid sinal secretion may result from decomposition of sinal secretion by secondary infection with saprophytes or gram-negative organisms or from primary infection by gram-negative organisms. This type of sinal secretion often is of dental origin and should not be radically treated by the rhinologist until after complete dental examination and treatment. Maxillary sinusitis of dental origin usually produces irreversible structural changes in the sinal mucosa in much less time than is required by primary sinal infection. However, certain patients with sinusitis of dental origin will recover completely after proper diagnosis and treatment of the dental disease even though the sinus had been involved for a considerable period.

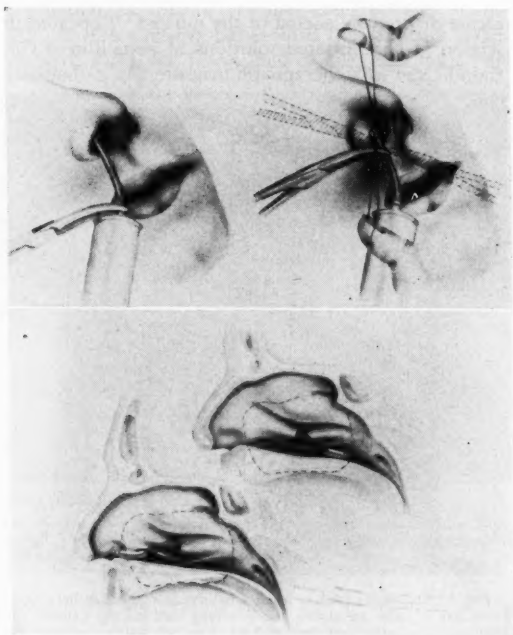

FIG. 4. Above. Diagram showing on the left the method of inflating the balloon with iodized oil and on the right the method of tying the end of the catheter. In grasping the catheter with the hemostat it is important to avoid rotating the catheter. This technique allows the knot to be tied on the side of the catheter away from the septum and forestalls the tendency of the knot to irritate the septum by pressure. To remove the balloon the iodized oil is aspirated with a small eustachian catheter. The balloon is then grasped near the antrotomy with a small hemostat and pulled out.

Below. Diagrams showing the two positions of the intranasal end of the catheter. The posterior position interferes less with respiration; however, it is not recommended until the balloon has become well distended because in certain patients the catheter may be difficult to retrieve for adding oil. In the anterior position the tip of the catheter or knot if not cared for properly may irritate slightly the vestibule or septum.

5. *Cough, chronic bronchitis, bronchiectasis, and pulmonary suppuration coexisting with purulent maxillary sinusitis.*—Cough is the most frequent symptom of suppurative sinal disease. During the early stages of sinusitis, cough is usually caused by drainage of purulent secretion from the sinuses into the trachea and bronchi. If cough persists after relief of drainage, usually infection and pathologic changes have occurred in the bronchi as a result of long-continued contact with the sinal secretion. Clinically, the cough and sinal drainage are closely related. During acute and subacute purulent maxillary sinusitis, cough may be a prominent symptom until the secretion in the sinus is evacuated by irrigation. Usually the cough is relieved immediately but it may return as soon as sufficient secretion is re-formed to drain into the larynx.

Apparently only a slight secretion from the sinuses can promote and continue the progressive changes in the tracheobronchial tree which are secondary to sinal disease. This phenomenon is evident after the Caldwell-Luc operation in which the clinical result is not perfect and a small amount of purulent sinal secretion continues to drain into the bronchi.

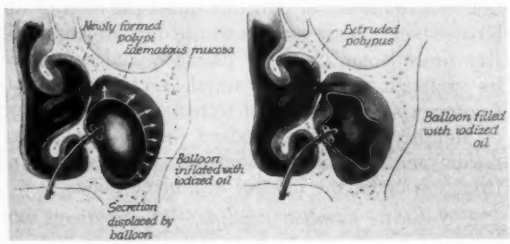

FIG. 5. Diagrams showing lines of force and results of pressure exerted by balloon upon edematous sinal mucosa. If the sinal mucosa is uniformly edematous, the balloon may force the mucosa in the form of newly formed polypi through the natural ostium, as seen on the left. This is similar to the formation of polypi in the middle meatus during acute maxillary sinusitis. On the right is seen the displacement of intrasinal polypi through the natural ostium into the middle meatus. As these polypi are prolapsed they may become strangulated and should be removed.

Application of local pressure to the mucosa of the maxillary sinus in patients with disease in the tracheobronchial tree often gives spectacular results. When the pressure is supplemented by bronchoscopic drainage, certain patients apparently have been cured or so greatly improved that lobectomy or segmental pneumonectomy can be avoided. Several of my pa-

tients have lost their productive cough, have gained weight, and have had partial or complete reversal of bronchial symptoms as evidenced by the clinical course of the disease and bronchography.

OPERATIVE TECHNIQUE

The application of local pressure to the mucosa of the maxillary sinus is accomplished by making a small opening in the nasoantral wall under the inferior turbinated bone. Through this opening into the maxillary sinus is inserted a rubber balloon which is formed around the self-retaining head of a 12 or 14 French rubber catheter. The head of the catheter may be selected to suit the size of the opening in the nasoantral wall; it should cover the antrotomy and prevent the balloon from herniating through the incision into the nasal cavity.

The balloon is expanded with iodized oil immediately after insertion or later until it fills the sinal cavity, displaces the secretion, and exerts gentle pressure on the sinal mucosa. As the secretion is displaced and the edematous fluid in the sinal mucosa is absorbed, additional oil is added at intervals of from one to three weeks. The balloon usually is left in the sinus for two or three months.

To avoid infection, an aqueous solution of sodium penicillin G, 1,000 units per cubic centimeter, is injected into the sinus immediately before the balloon is inserted. Also, penicillin is administered intramuscularly for a few days after the balloon is inserted.[4]

DIFFICULTIES IN APPLICATION

Difficulties which may be encountered in applying local pressure to the mucosa of the maxillary sinus are as follows:

1. *No cavity in the maxillary sinus.*—Roentgenologic examination with contrast media is essential to determine whether pressure should be applied to the mucosa of the maxillary sinus. Correlation of evidence from this examination with the sinal history is useful in deciding the probable antecedent pathologic processes and the immediate and future reaction of the sinal mucosa to the pressure treatment. Factors too numerous to mention here enter into the proper interpretation of the degree of cavitation in the maxillary sinus. The most important of these factors is differentiating between (1) the amount of edema of the sinal mucosa which is due to subacute infection and retention of irritating secretion and of secretion of high osmotic pressure and (2) the degree of thickening of the sinal mucosa which has resulted from more permanent pathologic processes.

Infected sinuses should be irrigated with sterile normal saline solution for a period to encourage formation of the mucoid secretion before a balloon is inserted into the sinus. These irrigations should not be supplemented by injection of any type of antibacterial agents, for too often these drugs tend to maintain or increase the edema. One of the most

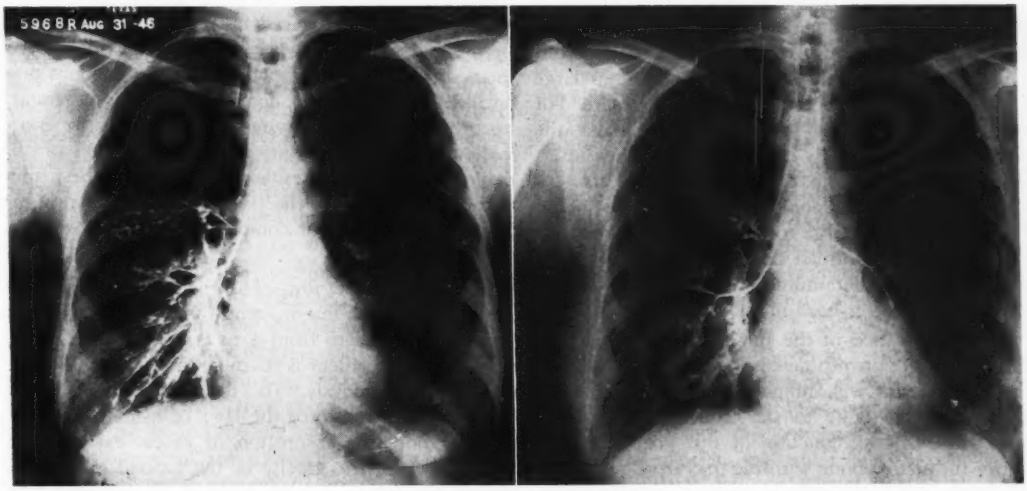

FIG. 6. Left. Bronchogram showing bronchiectasis involving the lower lobe of the right lung. This radiograph was taken in August, 1946. This patient Mrs. J. H. W., had had bronchoscopic drainage several times without appreciable improvement. Unilateral (left) maxillary sinusitis which she had had for fifteen years was then diagnosed, and pressure applied in the left maxillary sinus for three months resulted in complete cure of sinusitis. After additional bronchoscopic drainage, she lost her productive cough entirely and gained 20 pounds.

Right. Bronchogram of the patient on the left, taken in January, 1950. The patient was free of pulmonary and sinal symptoms and no pus could be found in the maxillary sinus or bronchi. The bronchogram shows some sacculation of the bronchus in the anterior basal segment but the areas do not contain pus. In this patient the progress of the bronchiectatic process apparently was arrested by removing the focus of drainage of purulent secretion from the sinuses by application of local pressure to the mucosa of the maxillary sinus.

iniquitous procedures advocated recently is the placing of indwelling needles in the maxillary sinus and the frequent instillation of concentrated solutions of penicillin. In certain patients this practice apparently has resulted in permanent thickening of the sinal mucosa and loss of motility of the cilia. Treatment before insertion of the balloon should be directed toward resolution of infection and creation of as much cavitation of the sinus as possible. If this resolution is not achieved, exacerbation of the infection or irritation of the mucosa by inserting the balloon into a small cavity may cause additional swelling of the mucosa and result in extrusion of the balloon into the nasal cavity.

2. *Antrotomy too large or improperly placed.*—Heretofore, the rhinologist in making an antrotomy under the inferior turbinated bone has been interested primarily in creating an opening almost as large as possible in an effort to prevent its closure. For treatment by a baloon in the maxillary sinus the antrotomy should be only large enough to allow insertion of the deformable catheter head and its surrounding collapsed balloon. Also, the opening should be only sufficiently large to allow it to be closed effectively by the re-formed catheter head. In some instances a balloon containing a 12, 14, or occasionally a 16 French catheter head may be required.

The ideal location of the antrotomy is in that portion of the inferior turbinated bone which forms part of the nasoantral wall immediately beneath the attachment of the bone to the lateral nasal wall or through the inferior junction of this portion of the inferior turbinated bone with the maxillary bone. The incision should be near the anteroposterior center of the medial sinal wall in this location so that the balloon may expand uniformly against the walls of the sinus. If the antrotomy is placed too far anteriorly or posteriorly, it may impinge against the anterior or posterior wall and produce pain before the balloon becomes effectively distended; this possibility is particularly important if the opening is placed too far anteriorly. The region of the nasal opening of the nasolacrimal duct is particularly susceptible to irritation and infection and should be carefully avoided.

3. *Herniation of the balloon.*—Herniation may occur through the natural ostium into the middle meatus; this is rare and usually occurs only in the patient with a large antral opening under the middle turbinated bone. The balloon, as it herniates into the middle meatus, may force intrasinal polypi into the middle meatus and strangle them. When strangulation occurs, the prolapsed polypi should be removed, care being taken not to grasp the herniated portion of the balloon and cause its evulsion.

Herniation of the balloon through the antrotomy under the inferior turbinated bone is one of the most frequent difficulties encountered. Usually it results from an opening which is too large or an insufficiently large head of the self-retaining catheter in the balloon. Also, if the entire balloon is not inserted into the sinus either because of insufficient sinal cavitation or an antrotomy which is too small, part of the balloon may remain in the nasal cavity and expand as the balloon is inflated with iodized oil. Herniation may be minimized by avoiding complete distention of the balloon immediately after insertion into the sinus. Also, visual observation of the region of the antrotomy during the primary and secondary inflation of the balloon is valuable to avoid herniation and application of too much pressure in the balloon. Herniation or prolapse of portions of the balloon into the nasal cavity in itself is not serious. Only if the efficiency of the balloon is reduced or if a troublesome nasal stenosis results should herniation be regarded as remarkable. In certain instances with the balloons now available commercially, some herniation is necessary to procure sufficient cavitation into which the entire balloon later may be inserted. If herniation of the balloon becomes a serious problem, the best procedure is to remove the balloon and wait until the antrotomy partially or completely closes before reinsertion.

4. *Incorrect pressure.*—Fluid is absorbed from the interstitial spaces intermittently until the tissue pressure is increased to from 4 to 8 cm. of water. After this pressure gradient is reached, interstitial fluids are absorbed continuously.[1] This means that little pressure is required to reinforce the laws of osmotic pressure and promote the absorption of excess extravascular fluid. The specific gravity of the iodized oil used to inflate the balloon is 1.35; the specific gravity of the secretion and tissue fluid is from 1.027 to 1.030, depending on the amount of protein contained. Therefore, as the balloon distends, the weight of the oil itself is sufficient to promote a certain degree of absorption. The balloon should not be expanded to a degree which produces discomfort or pain; the tolerance of the patient for the amount of pressure applied should be the chief criterion. After instillation of oil into the balloon, the patient should be observed for one hour to be sure that there is no discomfort of any degree in the sinus.

5. *Insufficient period of application of pressure.*—Most of my patients have worn the balloons for two or three months. Several have kept them in the sinus from four to six months. In certain patients with purulent sinusitis cure was obtained and in others good results have been reported after application of pressure for from two to four weeks. The longer period gives the best results because as the oil distends the balloon for months, it reduces the elasticity of the rubber and allows the balloon to conform to

the irregularities of the walls of the sinus, thereby exerting uniform pressure against all areas of the sinal mucosa. Certain patients, particularly those having allergic sinusitis with secondary infection and bronchial asthma, have worn balloons in their sinuses three or four times at intervals of a few weeks or months with complete relief of symptoms.[5]

6. *Infection.*—The maxillary sinus may become infected immediately after the balloon is inserted into the sinus or later may show signs of acute infection as a part of a respiratory infection. Infection may be minimized by instillation of penicillin into the sinus, 1,000 units per cubic centimeter, just before the balloon is inserted and by the intramuscular administration of penicillin for several days.

7. *Pain.*—The balloon should be removed any time pain or acute infection becomes an important symptom. By adhering closely to this principle, I have avoided any serious complications such as osteitis, osteomyelitis, periostitis, or pressure necrosis. Even though the balloon has been in the sinus several weeks, it should be removed and antibiotic therapy instituted if considerable pain or slight swelling of the face occurs. Pain and swelling is most likely to occur if the antrotomy is made too far anteriorly so that it interferes with the nasolacrimal duct. If the balloon is inserted and is greatly distended in this position, pain from pressure on the anterolateral sinal wall may occur. The patient should be instructed to clip the string around the end of the catheter if these symptoms appear at any time while the balloons are in the sinuses. Many patients are not conscious of the balloons. Pain resulting from irritation of the anterior part of the nasal septum by the tip of the catheter usually may be relieved by retying the end of the catheter and readjusting the position of the knot on the end of the catheter. Instead of leaving the end of the catheter at the mucocutaneous junction in the anterior nares, it may be turned toward the posterior nares; in this position it may be more comfortable and interfere less with respiration. This posterior position of the catheter is not recommended until the balloon has become well distended because in certain patients the catheter may be difficult to retrieve.

8. *Bad odor.*—Usually bad odor is caused by poor hygiene of the anterior nares and collection of crusts around the tip of the catheter and knot. This odor is effectively relieved by cleansing the tip of the catheter with hydrogen peroxide and lubricating the nares with white vaseline. Odor also may result from purulent secretion in the sinus which an incompletely inflated balloon fails to displace. The malodorous secretion usually occurs early after application of the balloon and often is due to underinflation to avoid herniation. This source of odor is relieved by more complete inflation.

SUMMARY

Increasing the tissue pressure by application of physical pressure to the surface of the part has been used for many years in numerous types of clinical pathology. Such treatment increases the tissue pressure, retards arteriocapillary filtration, and promotes venous absorption and lymph flow. If the pressure obliterates an air-containing cavity by means of an incompressible fluid medium, arterial pulsations are transmitted to the interstitial spaces and lymph vessels and help promote the flow of lymph and molecular diffusion. Time is important in the transfer of various molecules through the semipermeable membranes and tissue spaces of the body.

The sinal mucosa to perform its function must possess ciliary action, the ability to form mucus, and the ability to become edematous. The subacute stage of purulent maxillary sinusitis may exist for years before irreversible structural changes interfere with the nutrition of the sinal cilia and mucous glands. During purulent sinusitis, filling the cavity of the maxillary sinus with a balloon distended with iodized oil displaces the irritating sinal secretion, promotes absorption of edema fluid from the sinal mucosa, and allows the mucosa to return to normal or a semblance of normal.

The application of local pressure to the mucosa of the maxillary sinus frequently restores normal transillumination to the sinus. In certain patients the cure which results from this treatment is more complete and permanent than the end result obtained by the radical maxillary sinal or Caldwell-Luc operation; this factor is particularly important in those patients with cough, chronic bronchitis, and early bronchiectasis. Often in such patients only a small amount of postnasal sinal drainage will contribute to the activity of the bronchiectatic process. The first symptoms which disappear after the application of pressure are cough and excessive postnasal drainage. In selected patients bronchoscopic drainage as a supplement to the pressure treatment has been effective in avoiding pulmonary surgery.

Certain patients have been relieved of recurrent sinal infection, recurrent nasal polyposis, and bronchial asthma after the application of local pressure to the mucosa of the maxillary sinuses. Some of them continued to be free of these symptoms six years after treatment.

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ABSTRACT OF DISCUSSION

DR. JOHN H. BARRETT, Houston: My experience with the pressure treatment of maxillary sinusitis is limited. I first used Dr. Robison's procedure in cases in which the mucosa of the maxillary sinus was swollen and edematous due to allergy. It also has been my observation that the symptoms of secondary infection clear as the sinus mucosa is compressed by distention of the balloon.

This method of treatment has been used in a number of my patients with allergic sinusitis but in only 4 in whom allergy was not considered a factor. In all 4 the infection was subacute or chronic and the secretion secured from the sinus by irrigation remained mucoid or mucopurulent. Also, it was possible to clear the aggravating postnasal drainage, which was the chief complaint in each of the 4 patients. Two of them had severe cough and evidence of bronchopulmonary infection; the cough and bronchial symptom had subsided entirely in one but were still present in the other at the time of writing. The latter patient was the last treated by this method, and the balloon was removed only three weeks prior to last examination. At that time there was no longer purulent drainage from the sinus and the cavity was clear on transillumination. The cough and bronchial symptoms, while improved, had not entirely cleared, but there was hope that improvement would continue since the sinus disease had been eliminated.

Although the infection in all 4 patients was low grade, it could not be cleared with repeated irrigation. Perhaps an intranasal antrotomy would have been sufficient but some of my patients who have had an antrotomy still have purulent drainage from the sinus. So far none of the patients treated with pressure to the maxillary sinus have had to have a Caldwell-Luc operation; in some of my patients in whom antrotomy alone was used this procedure has been necessary.

I agree with Dr. Robison that pressure treatment is contraindicated in any acute infection, and I would not use it in a chronic infection in which the bone is involved or in which marked fibrotic changes in the sinal mucosa have occurred. In the latter type of patient I believe that the Caldwell-Luc operation is the treatment of choice.

Dr. Robison's advice that the antrotomy not be too large is important. If the opening is too large, keeping an even pressure in the sinus is difficult, as the balloon prolapses into the nasal cavity. Any deformity in the nasal septum in the region of the antrotomy I correct by a submucous resection. Only 1 of my patients had any pain, and this was caused by the stem of the balloon irritating a septal spur. All patients treated in this manner noticed an increase in the amount of postnasal drainage soon after the treatment was started but this drainage decreased as the treatment was continued. In 1 marked ethmoid infection cleared after maxillary infection subsided.

I am of the opinion that Dr. Robison's procedure offers a valuable method of treatment of maxillary sinus infection in which the changes in the mucosa are such that drainage alone is not sufficient. This pressure method should save certain patients from having a radical antrum operation and avoid the complications which occasionally follow this type of surgery.

DR. J. B. NAIL, Wichita Falls: I have been intensely interested, and many times baffled, in the treatment of chronic maxillary sinusitis.

Several years ago I reported to the Section on Eye, Ear, Nose, and Throat 100 consecutive patients upon whom the nasoantral window operation had been performed. As time went on, closure of the nasoantral window was observed in more instances than was pleasing.

In 1941 I presented a report of 85 consecutive patients upon whom the Caldwell-Luc operation had been performed. A sufficient number of cures were effected that I felt gratified, yet not wholly satisfied. Now, I think it extremely likely that a good many of these patients might have been spared the radical procedure and enjoyed equally as good results had they been treated according to Dr. Robison's procedure.

Following are case reports on 3 of my patients who were successfully treated by the method outlined by Dr. Robison:

CASE 1.—T. W. gave a history of chronic maxillary sinusitis for the five years prior to examination. The diagnosis was made by irrigation and roentgenography with Lipiodol. A stained preparation from the nose showed 3 per cent of eosinophils. Irrigation secured from both sides a chronic type of discharge which had a very bad odor. Dr. Robison's technique was performed and the balloons were removed from each side on the eighth day. The patient had had no recurrence of infection at last examination.

CASE 2.—D. C. gave a history of recurrent nasal polyps and a bilateral nasoantral operation twelve years prior to examination with recurrence and removal of polyps every two or three years in the interval. The diagnosis of chronic maxillary sinusitis was made by irrigation and Lipiodol injection with roentgenography. A nasal smear showed 4 per cent eosinophils. Balloons were implanted in each antrum; about four-fifths of each balloon was filled with oil and the remainder with air. This patient was operated upon three years prior to examination and had no recurrence afterward.

CASE 3.—F. S. gave a history of nasal obstruction and continuous drainage from both maxillary antrums for the seven years before examination. A nasal smear showed 50 per cent eosinophils. A large quantity of pus was irrigated from each antrum. A balloon implantation operation was performed and the balloons were removed after seven weeks. The patient had been symptom-free of allergy and sinusitis for the four years prior to last examination.

I have used the procedure outlined by Dr. Robison on 29 patients. The patients so treated were highly pleased with the end results and freedom from symptoms.

ANALGESIA AND EMOTIONS IN OBSTETRICS

The trend in modern obstetrics is to fit a pain-killing drug to the patient rather than the patient to a drug, states Dr. Curtis J. Lund, New Orleans, in the April 14 issue of the *Journal of the American Medical Association.*

Pointing out that apprehension and tension make labor more painful, Dr. Lund says that the need for analgesia can be reduced by proper attention to emotional factors in pregnancy and labor. Complications of pregnancy, he says, require individual attention. The average physician needs to know only two or three soundly established methods of analgesia fitted to the environment as one of these methods will usually be applicable to the patient.

RELATIONSHIP OF DENTAL PATHOLOGY TO DISEASES OF THE EAR, NOSE AND THROAT

M. P. SPEARMAN, M.D., F.A.C.S., El Paso, Texas

INFECTION or malformation of the teeth and jaws is rarely of local significance only. The status of the dental apparatus should be of deep concern to the otolaryngologist and ophthalmologist. Oral infections may lead to secondary manifestation elsewhere by (1) direct extension to adjoining parts, (2) hematogenous routes, or (3) the swallowing of infectious or toxic substances. Oral malformations may cause pain or other physiologic aberrations elsewhere by impingement on nerves supplying branches to adjacent organs.

CASE 1.—A. E., 52, a physician, complained of severe pain in the right ear. He was unable to use a stethoscope for long because of sharp pain in the ear radiating over the right mastoid area. He had been treated elsewhere for otitis externa with no relief.

The ear drums and canals were normal. Even though the patient was edentulous, dental consultation was insisted upon. Roentgenograms showed an infected, imbedded root of the third upper right molar; complete recovery followed its removal.

Inspection of the teeth will reveal only gross abnormalities. The physician is likely to be led astray by not insisting on roentgenologic examination and consultation with a well trained dentist; unfruitful experiences have taught me that not all dentists are helpful consultants.

CASE 2.—M. M., 23, a student, had been treated for "migraine" by her family physician for several years. Attacks required bed rest and at times opiates. The sinuses had been treated by a specialist for several years with nasal packs and irrigations of the antrum with no relief. Roentgenograms showed the sinuses to be clear; dental roentgenograms revealed an aberrant, unerupted tooth lying horizontally in the floor of the left nasal vault. Removal of the tooth resulted in complete relief of headache within three days. The patient was still free from so-called migraine seven years later.

Dramatic and gratifying results are often obtained by the physician who remembers his elementary training to look and think; a patient does not have sinus trouble just because he says so.

A rather common dental accident which deserves at least passing comment is described in the next case report.

CASE 3.—W. S., 52, a florist, complained of anosmia and nasal discharge after extraction of the first upper right molar one month previously. The dentist said that he had encountered unusual difficulty in removing the tooth, in that one root was extremely long and was infected at its tip.

Roentgenograms showed dense cloudiness of the right antrum. Antrotomy was performed and a small piece of thin bone, probably from the floor of the antrum, was removed. Recovery was rapid and complete.

Read before the Texas Society of Ophthalmology and Otolaryngology, Dallas, Texas, December 9, 1950.

Most physicians can recall many similar cases. The dentist should not be blamed for such an unfortunate occurrence; it is only remarkable that the floor of the antrum is not more often broken. Long, aberrant roots; infections; and a thin-walled cavity above contribute more in such mishaps than does unskilled dentistry.

CASE 4.—Mrs. D. S., 54, a housewife, complained of an extremely sore tongue. She had been examined thoroughly by a clinic. Blood counts were normal. No other physical disability was found. She had taken large quantities of vitamins with no relief.

The tongue was smooth, markedly hyperemic, and moderately swollen. The patient had used a certain brand of tooth powder for several years upon the advice of her dentist. Within three days after she had changed to a bland paste which was essentially soap and chalk, her tongue was well.

Certain dentifrices and mouthwashes contain astringents that may adversely affect the oral tissues; the most highly advertised of these seem to be the worst offenders. Pharyngitis, gingivitis, and glossitis of the most stubborn varieties often develop after their use. The cure is simple—to stop their use and change to a different brand.

DIZZINESS AND TINNITUS

Dizziness and tinnitus are two of the most troublesome symptoms which the physician encounters. Sometimes dramatic relief for patients so plagued can be obtained.

CASE 5.—Mrs. J. B., 35, a housewife, complained of dizziness and a low grade fever for six months. Her family physician had suspected undulant fever, tuberculosis, typhoid, and thyroid disease, among other diseases.

Tests and examinations yielded nothing; the sinuses, ears, and throat appeared normal. However, dental consultation revealed that the third lower right molar was infected. Removal resulted in prompt cessation of the dizziness and fever.

Certainly, tinnitus may be due to any one, or more, of a multitude of causes. Yet I have routinely considered dental disease first among the endogenous possibilities, many times with gratifying results to both the patient and me.

CASE 6.—M. L. C., 41, an accountant, had had a severe tinnitus for six months. He was referred to me after all systemic possibilities had been investigated by his family physician and several months of inflation of the eustachian tubes had failed to give relief.

My observations were noncontributory except for a marked imbalance in the bite. In addition, the dentist found several badly infected teeth which were removed; partial dentures were furnished to alter the bite. One month later all tinnitus had disappeared.

Complaints of vague pains in the face, scalp, neck, and shoulder may challenge the most astute diagnos-

tician. Many patients seeking relief from such symptoms will consult anyone and everyone who professes to practice the healing art. It is remarkable to note the role of dental imbalance in some such cases.

CASE 7.—B. P., 39, a waitress, had shooting pains and dull aching over the entire left face extending downward to the shoulder. She had undergone extensive examinations and treatments at the hands of several physicians and cultists with no relief.

My examination contributed nothing positive. A dental consultant reported no infection of the oral cavity. However, bite tests showed that the bite was about ten degrees off vertically. Suitable partial dentures corrected this imbalance and within one week all facial and cervical neuralgia had disappeared.

Sometimes I question the naive reliance upon the procedure of inflating the eustachian tubes, which is so misused that it might be called the "file 13" of the otolaryngologist. If all else fails and the physician is busy, it is convenient to tell the patient he has "blockage of the eustachian tube" and to inflate the tube forthwith.

CASE 8.—Mrs. C. W. M., 60, a housewife, had an annoying ringing and popping of the left ear for one year. She had been having the eustachian tube inflated twice weekly for several months with no relief.

The patient was edentulous. The dentist noted that the right side of her dentures were badly worn. New balanced dentures cured the ringing and popping of the ears within one week.

Chronic pharyngitis is an annoying, sometimes frightening affliction, and fear of cancer is a deep worry to many. Surely, patients with chronic pharyngitis need more than a hasty inspection, a swabbing, and a lozenge. Specific diseases, local and general, of course, must be sought. Again, dental infection should be a prominent consideration.

CASE 9.—Mrs. A. H. P., 66, a housewife, had had sore throat intermittently for one year. At times she had had extreme pain and difficulty in swallowing.

The pharynx was hyperemic and dry with streaks of bright red, lymphoid tissue. The tonsils were small. The anterior cervical nodes were tender bilaterally. The mucosa of the larynx was hyperemic and slightly edematous. There was a widespread gingivitis. Adequate dental care cleared the gum infection, the teeth were saved, and the pharyngitis disappeared.

Tic douloureux, or anything resembling this affliction, presents an undeniable need for help for its unfortunate victim. The agony of the patient when the trigger zone is stimulated is not pleasant to behold. Ticlike afflictions have long engaged the interest and sympathy of physicians. Many such cases are not true tic douloureux, as intelligent examination will demonstrate.

CASE 10.—Mrs. M. A. H., 42, a housewife, for several years had had intermittent, agonizing pains over the left face, ear, and head. She could initiate the attacks by touching the left temporomandibular region and had learned to shield that side of the face from drafts and stimuli of all types. She had taken all the anodynes known at one time or another and had consulted many physicians.

Examination was noncontributory except that dental opinion revealed the bite was several degrees off vertically. Several teeth were missing on the affected side and the opposite side had been used for chewing the food. Partial dentures restored the excursion of the jaws to normal and within a short time the pains about the face, ear, and neck had disappeared.

Almost fantastic is the amount of trouble some patients may experience because of dental disease. Some may enter a veritable maze of difficulties and wander up one blind path after another until rescued by the team of dentist and understanding otolaryngologist.

CASE 11.—A. L. R., 39, a clerk, noticed a diminution in hearing bilaterally for several years. At times he had had extreme dizziness and tinnitus. Twice weekly for a year and a half, he had been having the eustachian tubes inflated. Radium had been applied to the nasopharynx. Dramamine, nicotinic acid and vitamin B complex therapy afforded no relief.

When the patient consulted me, roentgenograms of the mastoid and skull were negative. There was an average loss of from 30 to 40 decibels in the right ear and 10 decibels in the left. Other eye, ear, nose, and throat findings were of no significance.

A neurosurgeon found no intracranial disease. The patient was referred for dental consultation because of a marked imbalance of the bite. It was necessary to remove all the teeth and to construct complete prostheses. When last seen, the patient reported that he was able to enjoy a beefsteak and symptoms had completely ceased. Recent audiograms have demonstrated no significant change in the hearing acuity, however.

The patient just described had spent much money and many miserable hours in the pursuit of health and comfort. This is not meant as adverse criticism at all, however, as often the correct procedure is clear to the consultant who enters the case after a number of needful examinations have been performed and adjudicated.

COMMENT AND SUMMARY

The role of foci of infection has been well debated in learned medical councils, hence it would be unfruitful to re-enter that domain at present. However, the otolaryngologist and ophthalmologist occupy a strategic position vis-à-vis dental infections and concomitant affections of the head and neck. So many vital and delicate structures are in the immediate neighborhood of the dental apparatus that it should be strikingly apparent that by their contiguity infections of the one may often and disturbingly involve the other. Physicians should cultivate a constant awareness of this fact to benefit those who consult them. Dental malformations and consequent mechanical disturbances of function may lead to alarming, annoying symptoms, just as does the more apparent offender, infection. Most dentists realize the importance of dental health, as do most physicians. The faults in both are apt to be forgetfulness and haste, rather than ignorance or neglect. It is incumbent upon physicians

and dentists to avoid assiduously the commission of any of these sins.

The hasty dental inspection for gross abnormalities and the opinion delivered to the patient on his next visit that "everything is all right" is to be avoided. Such an opinion is totally without value to the physician or patient. Stubborn insistence upon a complete study of the oral structures will often yield rich dividends. The otolaryngologist should soon learn to work with the dentist who is interested in more than stuffing cavities with metal.

The cases described herein contain little that is new but admonish the otolaryngologist to look, to understand, and to act.

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POTASSIUM DEPLETION: SYNDROME AND TREATMENT

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IN addition to sodium and chloride, potassium in recent years has been shown to be of great importance in therapy in certain altered states. Potassium is the major cation of the body, exceeding sodium by a ratio of more than two to one. It is the major cation of the intracellular space. Ninety-eight per cent of the potassium is contained here in the normal state, whereas sodium is the main cation of the extracellular space. Of the three ions, sodium, chloride, and potassium, only potassium seems to alter cellular function, owing to its specific ion effects.[7] In certain altered conditions potassium is lost from the intracellular compartment, and under these conditions it is replaced by sodium. If the deficit becomes great, the heart will be the chief organ to suffer as in the living organism it is particularly susceptible to prolonged harmful effects of potassium deficiency. If such occurs, there will result loss of conduction, necrosis of myocardium, cardiac dilatation, and failure.[7, 13, 22]

DEFICIENCY SYNDROME

Under what basic conditions may we expect to find a potassium deficiency?

Large amounts of potassium are contained in the gastric, biliary, and upper intestinal tract fluids, and prolonged loss of these fluids will result in potassium deficiency.[7, 14, 21] The kidney excretes from 8 to 10 Gm. of potassium daily. The kidney has no mechanism for conserving potassium as it does sodium, and even in states of severe potassium depletion, this cation continues to be lost in the urine.[7] The adrenal cortical hormone stimulates the excretion of potassium and the retention of sodium.[15, 16] In glycolysis there is an excess excretion of potassium.[11, 12, 19] In certain types of chronic nephritis, excess potassium is excreted.[11]

Thus, specifically, anything that causes excessive loss of gastrointestinal fluid over a prolonged period of time will cause a potassium depletion—prolonged vomiting, prolonged gastric and intestinal suction, biliary or intestinal fistulas, paralytic ileus, and severe infant vomiting or diarrhea. Since potassium continues to be lost in the urine even in depleted states, any condition that causes a loss of electrolytes with the patient's inability to partake of oral food over a period of time will result in potassium deficiency.[7, 9] Since the adrenal cortical hormone increases the excretion of potassium and retention of sodium, potassium depletion may be expected after adrenocortical response from severe trauma, shock, or anoxia of cells.[10, 17] This may be aggravated by the practice of some surgeons of administering adrenal cortical hormones for shock and severe trauma.[7, 20, 22] It may be expected in adrenal cortical tumors causing Cushing's disease or following the injection of ACTH or cortisone.[7, 15, 16] Potassium deficiency also may be expected in progressive inanition such as is seen in esophageal carcinoma.[9] Since potassium is lost during glycolysis, potassium deficiency may be expected in diabetic acidosis.[1, 18] The deposit of potassium in the intracellular space is enhanced by glycogenesis and the reverse is true in glycolysis.[7]

The majority of cases of potassium deficiency reported to date have been the result either of infant vomiting or diarrhea, prolonged gastric suction, intestinal fistulas, or diabetic coma.[6, 7, 8, 9, 13, 19, 21] The pathologic physiology seen in this deficient state is as follows: Loss of potassium occurs, resulting in depletion of this ion in the cells; sodium enters the cells as replacement of the potassium ion; potassium continues to be lost in the urine as the kidneys do not conserve potassium even during depleted states. Potassium deficiency is usually associated with salt and water loss. The symptoms of salt and water depletion are clinically evident, and replacement of these losses is commonly instituted without delay. With associated potassium depletion, there is an altered renal function with retention of sodium both in the intracellular and extracellular spaces with resulting alkalosis, which remains until potassium is made available. With continued potassium loss in the urine and with further replacement of this intracellular ion by sodium there is an alteration of cellular function which causes the

following signs and symptoms: abdominal distention with poor intestinal tone, nausea and vomiting, progressive muscle weakness, dyspnea with gasping respiration, cyanosis, bradycardia, extrasystoles or irregular rhythm, and cardiac decompensation.[7, 8, 9]

Several laboratory methods are useful in making diagnosis of potassium deficiency. It is well to remember that symptoms of this potassium deficient state are not evident, and laboratory tests are not indicative of potassium deficiency until after hydration has been established.[5, 7] In the potassium depleted patient, after salt and water replacement, one may expect a persistent carbon dioxide combining power of more than 60 cc. per 100 cc. of blood plasma, with low serum chloride levels, electrocardiographic findings of flattening or inversion and prolonged T waves, prolonged QT interval and possible extrasystoles or auriculoventricular block.[3, 7, 8] Blood potassium levels of less than 3.5 milliequivalents or 12 mg. per 100 cc. of serum indicate potassium deficiency.[7] Though laboratory findings are important in the diagnosis, more important is an intelligent evaluation of clinical states likely to be associated with this deficiency.

TREATMENT

Recognition of the conditions in which a potassium deficit is likely to occur is the most important factor for the treatment of such a deficit.[7, 9, 13] When the depletion is far advanced, five or six days sometimes will be required adequately to replace the potassium.[7] In patients expected to be carried on parenteral fluids four or more days, it is wise to use potassium prophylactically starting the second or third postoperative day, and it is well to impose a rigid limitation of sodium infusions to prevent not only edema but also potassium depletion since excessive sodium displaces potassium.[7]

The following general rules may be observed in the administration of potassium:

1. If possible, potassium should be given as potassium chloride, orally. Except in advanced renal disease there is no danger of potassium intoxication from this route.[6, 7] Doses up to 30 Gm. daily have been given by this method in equal parts of water and syrup of citric acid.[9] It also may be given in orange juice or milk.

2. The administration of potassium chloride by the subcutaneous route is safer than giving it intravenously; it is absorbed more slowly and there is less danger of intoxication. It has been recommended that the element be given in saline solution,[5] but usually when potassium therapy is indicated in adults, they already have received their tolerance of sodium chloride. A safe method of subcutaneous administration when saline solution is contraindicated is to give the potassium in 2.5 per cent glucose in water.

3. Potassium by the intravenous route is the third possible method of administration. Much has been written about the toxicity of potassium, chiefly affecting the heart, if given too rapidly by this route.[7, 9] Doubtless toxicity can occur, but even Darrow states he has never seen it.[7] I believe potassium can be administered safely by this route even in considerable quantities, provided it is definitely established that the patient is suffering from the potassium deficiency.

The solutions in which to give potassium, the amount of the initial dose, and the time period in which it is to be administered are not clearly understood; each author has different ideas on the subject.[2, 7, 9, 21] Darrow uses 2.7 Gm. of potassium chloride per liter of glucose and saline[7]; Burrows adds phosphorus to the solution.[4] Some authors use it in glucose and water.[8, 9] Some give it routinely, no more than 2.23 Gm. per liter,[21] whereas others have given as much as 6 Gm. in 500 cc. of solution.[2] Some advise giving potassium over a period of three to four hours,[7, 9] whereas others having given 7 Gm. in one hour.[2]

I believe it can be concluded that potassium may be given in any solution not hypotonic. Recently I have given it in Amigen. In cases of desperate potassium depletion, little danger of toxicity will be encountered if the element is given in relatively large amounts in a short time. In the usual surgical cases in which a potassium deficiency is to be anticipated, chiefly in patients who have lost large amounts of gastric or upper intestinal tract juices, the surgeon usually has replaced the electrolytes sodium and chloride, and I believe the conditions will only be aggravated by additional quantities of them. I believe potassium in these cases should be given in glucose with water as glucose will aid in the deposition of potassium in the cells. If the physician is supplying daily fluids in the form of Amigen, it may simply be added to the Amigen solution.

Potassium chloride comes in 20 cc. ampules containing 40 milliequivalents or 1.55 Gm. of potassium chloride. The amount desired may be injected into a solution bottle containing 500 or 1,000 cc. of fluid. Prophylactically, if the patient is given 3 to 4 Gm. per day, he should suffer no depletion. In the already depleted patient, 4 Gm. may be given initially over a period of about three hours. This dose may be repeated in four hours. It is wise in treating these patients to follow them with serial electrocardiograms and carbon dioxide combining power tests, and when the T waves and the carbon dioxide combining power have come back to normal, the patients may be carried on a maintenance dose of potassium.[9] It is well to remember that potassium never should be given except in those instances in which the patients are well hydrated and have well established kidney function. If these factors are not known, it will be wise to give an

infusion of from 600 to 800 cc. of glucose in water preceding the addition of potassium.[9]

Potassium therapy is contraindicated (1) in acute or chronic renal disease unless the serum potassium level is low and the indications are evident[7, 21]; (2) during or for twenty-four hours after major surgical procedures[7]; (3) in Addison's disease; (4) with severe burns in the acute phase; and (5) in acutely dehydrated patients until rehydration and adequate urinary output have been established.[21]

SUMMARY AND CONCLUSIONS

A brief consideration is given to the causes of potassium depletion; the pathologic physiology, symptomatology, and useful laboratory tests of aid in the diagnosis are summarized.

The most important aspect of management of this deficiency is the awareness of clinical states likely to be associated with it so that prophylactically, potassium may be administered to prevent an outright depleted state.

The physician should bear in mind the contraindications to potassium therapy. The most common of these is dehydration with inadequate urinary output.

When a definite diagnosis of potassium deficiency has been established, little danger is involved in the administration of potassium. It may be given intravenously in any solution commonly employed for intravenous use.

In surgical patients who suffer marked potassium depletion, it is wise to limit rigidly the intake of sodium chloride.

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STREPTOKINASE TO LIQUEFY BLOOD CLOTS

Use of two biologic compounds, streptokinase and streptodarnase, in liquefying blood clots, pus, and other waste products produced by certain types of infections and removing these products from the diseased area has been described in the March 3 issue of *The Journal of the American Medical Association*.

Drs. Joseph M. Miller, Milton Ginsberg, Raymond J. Lipin, and Perrin H. Long of Johns Hopkins University School of Medicine, Baltimore, reported excellent results in 80 of the 85 cases treated with streptokinase and streptodornase to remove waste material from infected and wounded areas. The doctors said the remaining 5 cases appeared to have advanced too far for the treatment to be effective.

Thirteen different kinds of infections were represented in the group treated. The compounds were found to be most effective in treatment of injuries characterized by clotting and the collection of pus and debris into walled off areas which, the J.A.M.A. article states, produce a honeycomb-like partition of the infected tissue. The compounds are injected by needle into closed areas and a nylon pack saturated with the substances is inserted into open infections and wounds. Walls of the honeycomb structure are dissolved and liquefied, making it possible for the waste materials to be drained.

EMOTIONS AND HEART DISEASE

Emotions can aggravate heart disease or cause psychosomatic symptoms resembling those of true heart ailments, thus complicating the doctor's job of diagnosis, according to Dr. Mark D. Altschule, author of an article in the March issue of *Circulation*, the journal of the American Heart Association.

Dr. Altschule claims the exact manner in which human feelings act upon the heart and blood circulation is for the most part unknown, and therefore the subject of emotional influences on the circulation is seen as a promising field for further scientific study.

CASE REPORTS

ATOPIC CATARACT: A CASE REPORT

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CATARACTS associated with different dermatologic conditions have been noted for some time. Rothmund in 1868 reported cataracts in several cases of telangiectasis, and Werner in 1904 reported 4 cases associated with scleroderma. Sack[4] in 1947 completely reviewed American, European, and Australian literature on atopic cataract, reporting on 30 cases in America and 28 in Europe and Australia. His own case report is the most recent, bringing the total to 62. This is a relatively small number, since atopic dermatitis (neurodermatitis) occurs not uncommonly, and one might expect that in any disease affecting the ectodermal layer the crystalline lens, being a portion of the surface ectoderm, would be involved.

In differentiating atopy from other forms of allergic sensitivity, Sulzberger[5] describes it as that form of allergy which appears to be peculiar to man, influenced by heredity, and characterized by the common occurrence of certain specific circulating antibodies, these antibodies being demonstrable by the Prausnitz and Küstner transfer experiment. Coca[2] has called these antibodies atopic reagins and has demonstrated their qualitative difference from precipitins. Sulzberger lists seven characterizing criteria: (1) symptoms and phenomena of increased vascular permeability, (2) a family history which is positive for various allergic manifestations, (3) a personal history that often reveals atopic manifestations other than the presenting disease, (4) eosinophilia in the blood smears, in the fluids and tissues of the affected parts, or in both, (5) positive reaction to skin tests and frequent demonstrability of passive transfer antibodies, (6) relief of symptoms on avoiding offending substances, and (7) recurrence or exacerbation on exposure.

Atopic dermatitis manifests itself first in infantile eczema, the patient commonly developing a papulovesicular rash which begins on the cheeks and soon extends to other parts of the body. The nature of the dermatitis changes as the patient grows older, representing a transition from the infantile to the adult type of sensitivity, and in childhood may present a combination of characteristics. At this stage papulation, rather than exudation and vesiculation, characterizes the skin condition.

Sulzberger[5] describes the outstanding symptom of the dermatosis in adulthood as itching, continuous or in crises, and the primary dermatologic lesion as a papule or a number of confluent papules which form lichenified areas. If the case be uncomplicated, there is no vesiculation, but external irritation or infection may cause exudation and crusting. The lichenified plaques are not sharply outlined but are usually surrounded by scattered, outlying papules. In typical cases the predominant localization is in the antecubital and popliteal spaces and the front and sides of the neck. While no skin area is immune, other favorite sites are the eyelids; the forehead and scalp; the perionychial areas and dorsa of the fingers; and the dorsa of the wrists, toes, and feet.

Cataracts in patients affected with neurodermatitis fall into two categories, described by Beetham:[1]

"...; the first is the typical and commonly seen complicated cataract. This usually begins at the posterior pole of the lens with increased iridescence, vacuoles and precipitates... Striation of the lens fibers is often present; small punctuate opacities are common, and iridescent crystals are frequent. These changes spread peripherally, and similar changes are soon noted in the anterior cortex, at the pole, immediately beneath the capsule. The changes progress, and the entire cortex becomes cloudy, forming an intumescent or a homogeneous mature cataract..."

The second type he describes as follows:

"...a dense, irregular plaque of opacity in the anterior cortex, occupying the pupillary area immediately beneath the capsule of the lens and possibly involving it to some extent. Several authors refer to these as anterior polar and anterior capsular opacities. The changes are entirely cortical, with possibly some wrinkling and irregular elevation of a normal capsule covering the opaque and distorted cortex."

These cataracts develop at an early age and mature rapidly.

Various theories as to the etiology of the cataracts have been advanced. Daniel[3] in 1935 attributed the cataract to interference with the metabolism of the lens and its transparency by a fine change in the function of the epithelial cells of the ciliary body, in the content of the aqueous fluid, or in the permeability and selectivity of the lens capsule.

Woods,[3] in discussing Daniel's report, stated that although he had never seen a patient with this particular syndrome, he had found her first two suggestions "meaningless... in relation to allergy." He held her last theory to be more acceptable in that the lens capsule, participating in the allergic reaction, might become permeable to normal aqueous, this filtering aqueous producing a precipitation of lens

protein and causing the formation of a cataract. "Such a mechanism would really be that of a traumatic cataract, allergy to the lens capsule being the traumatizing factor." However, Woods advanced objections even to this hypothesis, questioning the presence of specific allergens in the aqueous. It was at Woods's suggestion that Beetham did intradermal tests with aqueous removed from the eyes of 2 of his patients affected with atopic cataracts. The reaction in both cases was negative.

TABLE 1.—*Table of Positive Skin Tests Performed on G. M. at Balyeat Hay Fever and Asthma Clinic in June, 1949, and March, 1950.*

Substance	June, 1949	March, 1950
FOOD		
Wheat	1	0
Buckwheat	2	2
Corn	0	3
Egg—whole	2	0
Cheese	1	0
Coffee	1	3
Cocoa	0	3
Blackeyed peas	1	0
Peas	2	0
Asparagus	2	0
Spinach	2	2
Squash	1	0
Cucumber	1	0
Peach	0	2
Raisin	1	0
Soy beans	2	2
Okra	1	0
Beef	0	3
Chicken	1	0
Pecan	2	0
Cottonseed	2	4
Liver	3	2
Peanuts	3	2
Shrimp	3	
Oyster	2	
POLLENS (all 1:1,000)		
Giant ragweed	1	0
Western ragweed	2	0
Amaranthus retroflexus	1	1
Russian thistle	0	1
Bermuda	2	2
Johnson	2	2
Blue	1	0
Pyrethrum	2	0
ANIMAL DANDERS (all 1:1,000)		
Duck feathers	0	2
Goose feathers	1	0
Cattle hair	0	2
Rabbit	0	1
Cat hair	1	0
Dog hair	1	0
House dust	2	2
Elevator dust	1	
Orris root	2	0
MOLDS (all 1:1,000)		
Alternaria	3	
Aspergillus	3	
Cephalosporium	1	
Fusarium	0	
Trichophyton	1	
Hormodendrum	0	
Monilia albicans	2	
Mucor	2	
Penicillium	2	

The cataract associated with atopic dermatitis is similar to that of known endocrine origin in the early age of onset, the rapidity of maturation, and the frequent bilateral involvement. However, no significant disturbance in the endocrine systems of the patients reported upon has been found.

Beetham[1] discussed a "universal tendency" toward malnutrition in the 10 cases which he reported in 1940, finding the patients to be from 10 to 25 per cent below normal in weight. He also considered avitaminoses but did not advance either malnutrition or

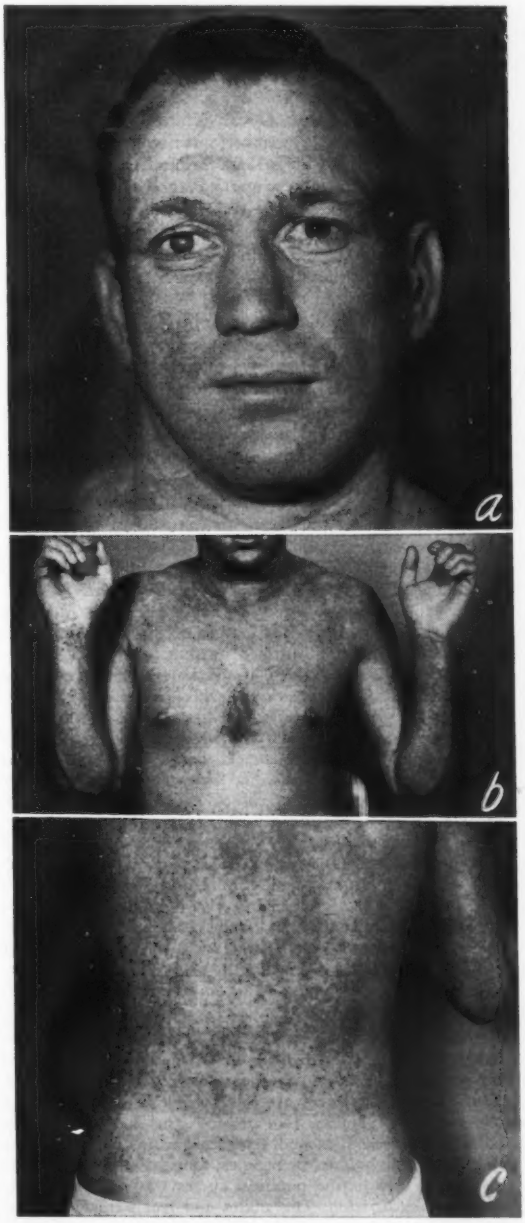

FIG. 1a. Photograph showing skin lesions of the face, a mature cataract of the right eye, and the left eye which has been operated upon.

b. Photograph demonstrating skin lesions of the forearms and chest.

c. Photograph revealing the appearance of the skin of the back.

avitaminosis as a causative factor, rather favoring the allergy as an explanation.

CASE REPORT

G. M., a 25 year old man, was first seen November 16, 1948, complaining of poor vision in both eyes. He gave a history of eczema, asthma, and allergy of long standing.

History.—The patient first developed eczema of the face and neck in the spring of 1923, when he was 3 weeks old. This condition was cleared with ichthyol therapy but recurred the following September, involving the antecubital regions, the popliteal areas, and the neck and cheeks. The rash persisted during the winter months but cleared during the summer. The next fall the skin again became involved in a like manner and did not respond to any local medication.

During childhood and adolescence the patient continued to have a rash in the areas of flexion of the knees and elbows, usually worse in the summer. In 1941, when 18 years of age, he had a severe attack of dermatitis involving most of the body; since then he has had repeated attacks of increasing severity. In 1947 the dermatitis became so severe that he had to be hospitalized for about two months. In the intervals between severe attacks the skin at times would become almost clear. The skin rash was always accompanied by severe itching. Sometimes the attacks followed periods of emotional strain, excitability, or change in temperature, while at other times no definite contributing factor was evident.

The treatment for the dermatitis consisted of various local applications such as lotions, ointments, baths, and sunlight; later, desensitizing injections and antihistaminic drugs were used, none of which proved to be very beneficial.

The patient gave a history of being allergic to different foods, dust, and pollens. The nature of the allergens varied from time to time as illustrated by skin tests June 9, 1949, and March 28, 1950, given at the Balyeat Hay Fever and Asthma Clinic, Oklahoma City (table 1) under the supervision of Dr. Dick H. Huff. The first attack of asthma occurred at the age of 3 years and the patient had continued to have asthma since, the attacks being worse during the winter. One paternal cousin had infantile eczema. The patient's mother has mild attacks of hay fever and one maternal aunt has hay fever.

The first history of any involvement of the eyes dated back to 1945, when the patient had an attack of iritis in the right eye which responded to atropine and hot compresses. In 1946 he became aware of blurred vision of the right eye and was told that he had an early cataract. About a year later the vision of his left eye began failing and a diagnosis of cataract in this eye was made. After that the vision of both eyes grew progressively worse, the loss being more gradual in the right.

Examination.—The patient, when seen by me in the fall of 1948, was a well developed, well nourished man who appeared somewhat older than the stated age of 25. The dermatitis was fairly generalized. The skin of the dorsum of the hands, wrists, and forearms was somewhat thickened and scaly and some papules were present. The skin of the neck and face appeared thickened, tense, and scaly. The trunk was fairly uniformly covered by papules, some of which were confluent. The skin of the popliteal spaces was also thickened, with some excoriation. The ankles and feet were fairly free of any skin involvement. (See figure 1.)

The vision of the right eye was 20/100 and in the left eye 20/100+. The vision of the right eye could be improved to 20/60 with the correction of —1.75 sphere, —0.75 cylinder, axis 90, and the vision of the left eye could be improved to 20/40 with the correction of —1.75 sphere, —0.50 cylinder, axis 90.

External examination of the eyes revealed some thickening of the skin of the upper and lower lids and thickened, reddened conjunctiva of the lids. The anterior chambers were normal in depth. The pupils reacted to light and accommodation equally well. The tension in the right eye was 21 mm. and in the left eye 22 mm. with the McLean tonometer.

Ophthalmoscopic examination showed an immature cataract in both eyes, with dense, centrally located opacities. The fundi were seen indistinctly.

Slit lamp examination of the right eye revealed the cornea and anterior chamber to be clear. There was a large clump of iris pigment on the anterior capsule of the lens. There was a large, dense, central star-shaped white plaque which appeared to be in the anterior cortex just beneath the capsule, and from this area opacities radiated toward the periphery. The nucleus appeared clear. There were rather dense posterior cortical opacities. In the left eye the cornea was clear and there were no deposits on the posterior surface of the cornea nor on the anterior surface of the lens. The anterior chamber was clear. Changes in the left lens appeared like those in the right lens but were not as marked.

In the spring of 1949, the patient was found to have glomerulonephritis. The only recent illness other than the dermatitis and asthma had been a mild infection of the upper respiratory tract during December, 1948. He had not been taking any sulfonamides. His blood pressure was somewhat elevated but there was no edema of the ankles or feet. Treatment for the nephritis consisted of bed rest and a high protein, salt-free diet.

Laboratory Studies.—Frequent laboratory checks were made. Early laboratory findings were similar to those on June 6, 1949, which were as follows:

The red blood cells numbered 4,750,000 per cubic millimeter; the hemoglobin was 97 per cent. There were 9,800 white blood cells per cubic millimeter with a differential of 1 per cent stab cells, 52 per cent segmented cells, 37 per cent lymphocytes, and 10 per cent polymorphonuclear eosinophil leukocytes.

The nonprotein nitrogen was 42 mg. and the urea nitrogen 33 mg. per 100 cc. of blood. The total serum protein was 5.3 Gm. per 100 cc. of serum; the albumin-globulin ratio was 1 to 1, with 2.65 Gm. of albumin and 2.65 Gm. of globulin per 100 cc. of serum. The sedimentation rate in 30 minutes was 45 mm. and in 60 minutes, 97 mm.

The urine had a specific gravity of 1.015 and an acid reaction. The albumin was 3 plus and sugar negative. An occasional white blood cell was noted.

On November 9, 1949, the laboratory tests were as follows:

Wassermann and Kolmer tests were negative. The nonprotein nitrogen was 28.5 mg. per 100 cc. of whole blood. The total serum protein was 9.44 Gm. per 100 cc. of blood. The albumin was 7.4 Gm. and the globulin 2.04 Gm. per 100 cc. of blood, an albumin-globulin ratio of 3.1 to 1.

The urine was yellow, cloudy, acid in reaction, and had a specific gravity of 1.029. The albumin was 4 plus and the sugar and bile were negative. One or 2 leukocytes, a rare red blood cell, a few flat epithelial cells, and a few granular and hyaline casts were noted per high power field. No bacteria or crystals were seen.

Surgery.—By December, 1949, the patient's general condition had improved sufficiently to allow surgery. The cataracts were of the intumescent type and the vision had been reduced to light perception for several months. On December 6, the patient had a combined extracapsular cataract extraction of the left eye. For two days following surgery he had a severe flare-up of dermatitis but there was no undue reaction in the eye which had been operated upon. There re-

mained some rather dense lens substance in the pupillary area, and a discission of the secondary membrane was performed March 14, 1950.

At last examination the eye was quiet, and with a correction of +10.25 sphere, +2.25 cylinder, axis 15, the vision was 20/25+.

CONCLUSION

The case presented is typical of atopic dermatitis with cataract formation, complicated by glomerulonephritis. There is no definite evidence as to the etiologic factor in the formation of these cataracts but it is reasonable to suppose that the allergy played an important part.

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ABSTRACT OF DISCUSSION

DR. REX C. HOUSE, Corpus Christi: Dr. Zanek's report of a case of atopic cataract represents a valuable contribution to the classification of cataracts of dermatogenous origin (also classified as cataracts associated with certain skin diseases).

Dermatogenous cataracts have been reported in association with the following generalized chronic skin diseases: (1) neurodermatitis, (2) poikiloderma atrophicans vasculare, (3) scleroderma, and (4) generalized eczema. These cataracts occur more frequently in women than men and usually develop during the third decade of life. A history of a chronic eczema preceding the cataract by a decade or longer is given. Dermatogenous cataracts usually develop in a few weeks; they may be unilateral but more frequently are bilateral.

The case presented by Dr. Zanek seems to me to fall into the classification of dermatogenous cataracts associated with neurodermatitis.

The etiology of cataract neurodermatosa has been given as: (1) pluriglandular imbalance (involvement of the thyroid, gonads, or pituitary glands), (2) metabolic imbalance, (3) nervous instability, and (4) allergy. None of these factors has been proved satisfactorily to be the cause.

Embryologically, the crystalline lens is of surface ectodermal origin, and it is logical to consider involvement of the lens in association with chronic diseases of the skin.

The typical change in the crystalline lens in dermatogenous cataract consists of a characteristic plaque, shield-like or saucerlike opacity in the anterior or posterior cortex with a clear nucleus, though a total cataract may develop. In view of the location of the lens opacities, it is my opinion that an intracapsular extraction is indicated.

Recently I had occasion to operate upon a patient in whom there were bilateral saucer-like central opacities in the posterior cortex; a history of multiple allergic manifestations and nervous instability was given. Thus, the cataracts were probably of dermatogenous origin. Intracapsular extraction was done without great difficulty with good visual results.

HYPERTHYROIDISM PRODUCED BY ORALLY ADMINISTERED THYROID EXTRACT

Report of Case

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THE concept that hyperthyroidism in all its manifestations can be produced by thyroid extract is not new. As early as 1906 Stegman reported on this phenomenon, and since then numerous authors have dealt with it. Brain[2] published a series of 20 cases in 1936, and two series, one consisting of 9 cases and one of 15, appeared in the Danish literature in 1945.[3, 5]

It should be emphasized that these patients did not have transitory episodes of hyperthyroidism which ceased when the hormone was discontinued. All cases reported were true Graves's disease characterized by increased basal metabolic rate, tremor, exophthalmus, tachycardia, and loss of weight but with or without goiter. Specific antithyroid therapy identical with that used in idiopathic Graves's disease (Lugol's solution, propylthiouracil, deep roentgen therapy, and surgery) was required to control the condition. All patients had previously been normal insofar as the thyroid status was concerned and they had been receiving oral thyroid extract for reduction of weight, sterility, dysmenorrhea, or "as a tonic." The dosages varied greatly: some patients had received as little as 1 or 2 grains daily; others had been taking relatively enormous amounts which ranged from 25 to 50 grains per day, with a few taking larger amounts.

In Stegman's original case it is interesting to note that the symptoms of hyperthyroidism did not appear until after thyroid medication had been discontinued. He wrote, "After thyroid treatment, the patient developed palpitations, and this led to the discontinuation of the treatment. During the next few months tachycardia, tremor, diarrhea, and loss of weight became conspicuous, and later exophthalmus appeared."

He reported that the patient's condition improved greatly after deep roentgen treatment.

Behrman[1] presented a similar experience of one of his own patients, stating, "It would seem that the administration of thyroid extract in a certain number of patients initiates a condition closely resembling, if not identical with, exophthalmic goiter."

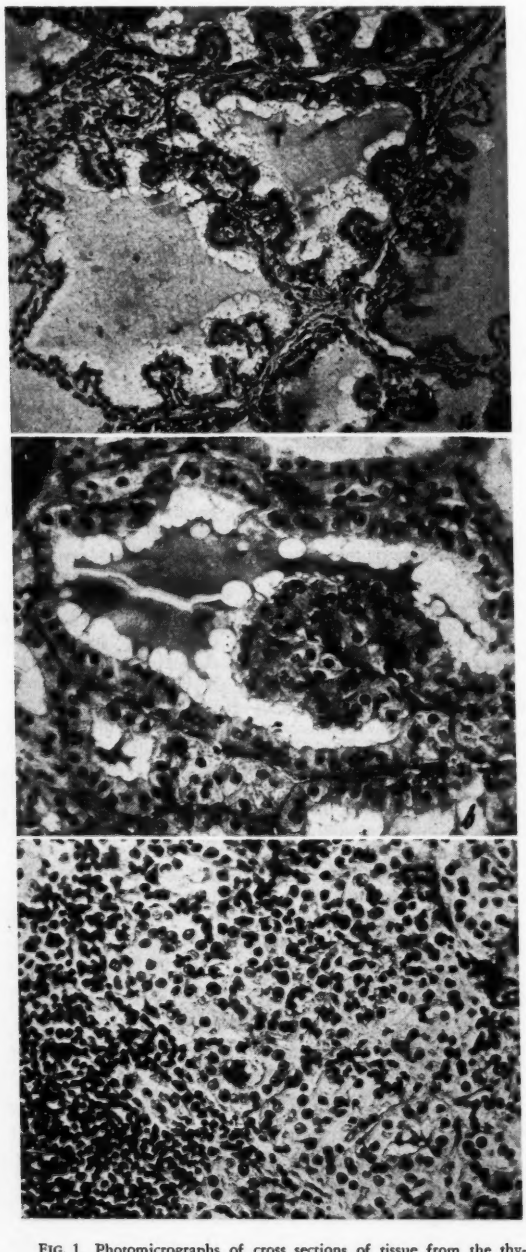

FIG. 1. Photomicrographs of cross sections of tissue from the thyroid gland of Mrs. B., in which the three criteria of Graves's disease are well shown, namely (a) epithelial hyperplasia and hypertrophy, (b) lymphoid hyperplasia, and (c) disappearance of colloid.

It is difficult to say what percentage of patients receiving thyroid extract for one reason or another develop thyrotoxicosis as a definite clinical entity. In one series extending from January, 1940, to September, 1944, 3.1 per cent of the total patients with thyrotoxicosis had developed the disease after taking thyroid. Of these 70 per cent had been getting the medication for simple obesity. All of the patients in this series were women, and most of them were in the climacteric age group.[3]

Another view, somewhat more radical, is held by Curtis,[4] who stated, "It is my experience that large doses of desiccated thyroid (10 to 20 grains daily) are quite unwarranted; personally, I never use them. I find that they uniformly produce hyperthyroidism which may be so 'masked' that the patient reveals the toxicity without the clinical picture of induced hyperthyroidism."

It is apparent that Graves's disease is not a common sequela of thyroid medication; nevertheless, it has been estimated that approximately five times as much thyroid extract is sold as would be required to treat all existing cases of myxedema. Why do so few patients who take thyroid develop thyrotoxicosis? Bruun[3] believed that the answer lies in the fact that a predisposition to thyrotoxicosis seems to exist in certain persons. He stated that this predisposition can be demonstrated in 45 per cent of patients in whom thyroid disease follows thyroid therapy.

CASE REPORT

Having established the fact that thyrotoxicosis resulting from thyroid medication is a well-recognized entity, with numerous authenticated cases appearing in the literature, the following case is herewith reported:

Mrs. B., a 34 year old, married, white woman, was first seen in August, 1950. Her complaints were increasing nervousness, irritability, palpitations, sleeplessness, and progressive bulging of the eyes. She stated that she had become so irritable she could no longer get along with her co-workers and was in danger of losing her job. Her speech was rapid and intense, and she burst into tears several times during the interview.

The history revealed that until six months prior to examination the patient had been taking 25 grains of thyroid extract daily to lose weight under a physician's supervision. At the beginning of treatment she weighed 212 pounds; in ten months she lost 32 pounds. She made frequent visits to her doctor during this period, and it was under his orders that the dosage of thyroid extract was gradually increased to the previously stated level. The total period during which the patient took thyroid was one and one-half years, but it was only for the final ten months that she took 25 grains daily. At the end of this time the patient discontinued the medication because "she had become extremely nervous and unable to sleep." Shortly afterward she noted uncontrollable tremor of the hands and short episodes of tachycardia.

Exophthalmus began to develop and the tremor, tachycardia, and nervousness became more conspicuous. Thus, the classic picture of hyperthyroidism continued to develop during the six months after discontinuance of the medication.

Physical examination revealed a highly nervous, emotional, moderately obese woman who spoke in a rapid-fire manner. Her movements were abrupt and somewhat spasmodic. The temperature was 100.1 F.; the pulse rate was 120. A marked bilateral exophthalmus was noted. The thyroid gland was diffusely enlarged to a moderate extent with no palpable nodularity. A fine bilateral manual tremor was evident.

Laboratory Data.—The basal metabolic rate was plus 34. The blood cholesterol was 200 mg. per 100 cc. of plasma. Other laboratory work was negative.

Course of Disease.—The patient was placed on appropriate antithyroid medication with Lugol's solution and propylthiouracil and, initially, a mild sedative. Results of this preparatory treatment were good. The basal metabolic rate came down steadily, the pulse dropped to 80, the tremor improved, and the episodes of tachycardia ceased. During treatment little change in the exophthalmus was noted except that it did not increase. The thyroid gland slowly enlarged.

Eight weeks after the onset of treatment the patient's basal metabolic rate was plus 1 and the pulse 75. She was anesthetized in her room and taken to surgery, and a bilateral subtotal thyroidectomy was performed without incident. The postoperative course was smooth and the patient was released from the hospital on the fifth day after surgery.

Two months after surgery Mrs. B. was virtually symptom-free; she was on no medication. The tremor, rapid pulse, and nervousness had disappeared and she was working in complete harmony with her fellow employees. The exophthalmus had definitely improved but not so much as would be desirable.

SUMMARY AND CONCLUSIONS

A brief study of the possible production of hyperthyroidism by the administration of thyroid extract is presented. A portion of the published material on this subject is cited and 1 case in point is reported.

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MANAGEMENT OF NEWBORN INFANT WITH SYMPTOMS INDICATIVE OF MYASTHENIA GRAVIS

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THE symptoms of myasthenia gravis in the newborn period are more likely to occur in the offspring of a mother suffering from the disorder than to develop spontaneously. Only 4 newborn infants with evidence of the disorder have been described[7, 12, 13] and all were born to mothers who had myasthenia gravis. On the other hand, many normal babies have been born to mothers afflicted with this condition.[3, 4, 5, 6, 8, 10, 14, 16] Important, however, is the fact that if myasthenic symptoms arise in the infant, death is likely to result unless intensive and adequate therapy is instituted.[12, 17] The successful treatment with neostigmine of an infant during the neonatal period seems sufficiently significant to warrant detailed presentation of the anamnesis.

CASE REPORT

A full term female infant was delivered spontaneously April 19, 1949, to a 30 year old Negro mother who had symptoms of myasthenia gravis of ten months' duration. This mother was reported on by Stone and Rider,[11] who mentioned the critical state of the infant. Because of the unusual situation, the newborn infant was scrutinized by members of the pediatric staff and nursing service. At delivery she appeared listless and inactive.

The birth weight was 2,500 Gm. and the length was 47 cm. The circumference of the head was 30.5 cm. and of the chest 30.2 cm. The color was good and the Moro embrace reflex positive. A weak cry and a questionable "ironed out" expression of the face were the most significant findings.

For the first three days the child seemed to do fairly well although the nurses reported that she refused to take nourishment from the bottle. When a medicine dropper was used for feeding, milk was regurgitated through the nostrils. By the third day, the face had become unmistakably mask-like (fig. 1a) and the cry was so weak it was almost inaudible. The mouth constantly remained in an open, relaxed condition and the facial features remained unchanged even during painful stimulation. From figure 1b, which was obtained when the infant was stimulated to cry, it is clear that no "lines of expression" were visible. The child allowed her lower jaw to drop, and her mouth gaped constantly. At this time, however, the muscle tone in the arms and legs seemed fairly good.

As a therapeutic test, at 4 p. m., sixty-eight hours after birth, the infant was given 0.07 mg. of neostigmine methyl-

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sulfate intramuscularly. Within ten minutes there was a dramatic response. The child began to cry lustily (figure 1c). When offered a nursing bottle, she sucked vigorously and the feeding was retained.

After this gratifying experimental period, a regimen of regular feedings plus 2 mg. of neostigmine bromide administered orally every three hours was instituted. The drug in an aqueous solution was added to the milk mixture.

Because of several bouts of loose stools during the second twenty-four hours of the regimen, the dose was lowered to 1 mg. every three hours. The infant remained entirely asymptomatic. By the eighth day of life, the birth weight had been regained. The infant continued to do so well that on the seventeenth day the neostigmine was withdrawn. No adverse effect was noted. The baby continued to take all feedings and gained weight so that by the time of discharge on the thirty-third hospital day she weighed 3,200 Gm. She showed no evidence of myasthenia gravis when she was seven months old. Five siblings of the child are living and well.

FIG. 1. B. W., a 68 hour old Negro infant born to a mother with myasthenia gravis.
a. Relaxed, expressionless face during rest.
b. Reaction when stimulated to cry before the administration of neostigmine.
c. Reaction ten minutes after intramuscular injection of 0.07 mg. of the drug.

DISCUSSION

From the study of this infant, as well as other similar patients, one may speculate that these newborn infants do not have true myasthenia gravis but suffer symptoms because of the carry-over of some humoral factor from the mother. Eventually this factor is neutralized or eliminated. Difficult to explain, however, if this theory were true, would be the infrequent occurrence of symptoms in infants of mothers with definite myasthenia gravis.

Although the underlying cause of myasthenia gravis is not known, evidence suggests that a circulating factor exists. In 1935 Walker[15] occluded the circulation in the upper limbs of such a patient and exercised the muscles of the forearm. When the constriction was released after a short latent period, the clinical signs of myasthenia gravis increased. Wilson and Stoner[17] in 1944 obtained essentially the same results. They also noted that the serum of myasthenic patients contained a substance which interfered with transmission of an impulse in muscle nerve preparations. Further evidence has been presented by Torda and Wolf,[13] who demonstrated that acetylcholine synthesis in mixtures of frog brain and serum of these patients was less than in control subjects. The more severe the myasthenia, the less acetylcholine synthesis occurred. Other methods which have been used to study the possibility of a circulating factor have yielded negative results. For example, Cohen[2] observed that the serum from a patient with myasthenia gravis had no effect on frog or leech muscle preparations.

The rarity of the disorder is evident in that only 4 newborn infants with myasthenic symptoms heretofore have been recognized and all were offspring of mothers with the disease. Bowman,[1] also Levin,[9] cited instances of children from 1 to 4 years of age, in whom, according to the history, the disease appeared to have developed spontaneously during the neonatal period. The mothers of these infants did not have myasthenia gravis. Several other observers have reported the onset to have occurred during infancy but not during the neonatal period.

The gravity of the prognosis is indicated by the death of 2 of the 4 newborn infants. The infant described by Wilson and Stoner[17] showed marked myasthenic symptoms practically at the time of birth and died on the fourth day of life. No specific treatment had been instituted. Strickroot, Schaeffer, and

Berge[12] stated that the infant on whom they reported at first apparently responded well to neostigmine but died after discontinuance of the drug on the sixth day of life. The attempt to reinstitute the drug shortly before death gave only temporary response. Incidentally, no tumor of the thymus was reported in the autopsy on this patient. The third infant, who was also described by Wilson and Stoner,[17] recovered but no cholinesterase inhibitors were used. For three or four weeks this infant exhibited limited respiratory excursions, cyanosis, and failure to gain weight. Sucking and swallowing were poor, and milk was regurgitated through the nose. LaBranche and Jefferson[7] recently reported their observations on a newborn infant with the disorder in whom the symptoms recurred when neostigmine was discontinued. The drug was still being used when the infant was 4 months of age.

The remarkable initial reaction to neostigmine as well as the continuous favorable response in the infant in our case report seemed to indicate that this was the only instance, with the possible exception of the LaBranche and Jefferson case, wherein adequate therapy was given.

In recent years the more satisfactory management of adult patients with myasthenia gravis undoubtedly has enhanced the likelihood of the birth of infants with the syndrome. On the basis of the available information, the recommendation that treatment with neostigmine be instituted as soon as myasthenic symptoms are suspected is justified. Because of the gratifying results obtained in our patient, it seems well first to give intramuscularly from 0.07 to 0.1 mg. of neostigmine methylsulfate for diagnostic purposes. If there is improvement, 2 mg. of neostigmine bromide may be given orally every three hours; it may be administered conveniently in aqueous solution by adding the drug to the regular milk feedings. This dosage may be more than the infant can tolerate; hence one should be alert to the development of diarrhea as a manifestation of overdosage. Under such conditions, it is advisable to lower the dose to 1 mg. but the administration should be continued at intervals of three hours.

Inasmuch as Strickroot, Schaeffer, and Berge discontinued the drug after two days with subsequent lethal outcome within a few hours, it seems particularly important to suggest that treatment be continued at least for about three weeks. Of course, the duration of treatment depends upon clinical judgment in relation to the manifestations.

There have been no reports concerning the use of tetra-ethyl pyrophosphate for myasthenia gravis in the childhood group. Because of satisfactory results obtained with the drug in adult subjects, it should be borne in mind that this organic compound may be used in the event resistance or undue tolerance to neostigmine is encountered. The condition of the mother of the child on whom we have reported was controlled well for six months with tetra-ethyl pyrophospate, whereas neostigmine previously had been unsatisfactory.

SUMMARY

The occurrence of symptoms indicative of myasthenia gravis in a newborn infant, with excellent response to neostigmine administration, is described. On only four occasions previously has a clinical picture such as this been recognized and recorded. All of the infants were born to mothers with myasthenia gravis. Two infants died and 2 survived.

The following conclusions regarding the management of such infants are made:

1. Infants born to mothers with myasthenia gravis should be observed closely during the entire neonatal period for symptoms of the disease.

2. If such symptoms appear, immediate intramuscular administration of neostigmine is recommended as a therapeutic test and, provided a satisfactory response is obtained, treatment with the drug should be continued for at least three weeks.

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ABSTRACT OF DISCUSSION

DR. CHARLES T. STONE, Galveston: It is not surprising that so few infants with symptoms of myasthenia gravis are born of mothers with the disease. Viets has estimated that there are only about 1,500 patients with myasthenia gravis in the United States, and since the disease occurs in all age groups and about equally in the two sexes, of necessity a small number of infants born of such patients are affected.

The available information does not make it possible to explain what happens in cases like that reported. The question naturally arises as to why the infants of most mothers with myasthenia gravis are born without signs or symptoms of the disease, whereas in an exceptionally small number the offspring may show for a time the classic picture of myasthenia gravis. These symptoms cannot be considered to represent a hereditary disease, since if adequate therapy is instituted promptly and maintained sufficiently long, recovery may ensue without any residua whatever.

Few cases of permanent remission of the disease in adults are noted; yet the evidence, scant though it is, gives the impression that infants born of myasthenic mothers who present the picture of the disease at or shortly after birth do so only temporarily. If they can be carried over this temporary period, recovery is usually complete.

Despite the absence of confirmatory evidence from the laboratory, it seems reasonable to hypothesize that the same disturbance in the acetylcholine-cholinesterase enzyme system which accounts for the symptoms in the mother is transferred to the infant through the placental circulation. Apparently this abnormality persists only as long as is required for the newborn infant to develop its own normal acetylcholine-cholinesterase balance. When this occurs, treatment may be discontinued but, as was well brought out in the paper, to omit therapy prematurely is to invite disaster.

My belief is that neostigmine is the remedy of choice. Tetra-ethyl pyrophosphate, which has been observed to be so effective in the management of adults with myasthenia gravis, cannot be given in full therapeutic doses immediately, and its physiologic action develops somewhat more gradually than that of neostigmine. The dose must be increased gradually. Since such a narrow margin exists between therapeutic and toxic levels of tetra-ethyl pyrophosphate, I fear that the drug might produce serious toxic effects in an infant in whom early recognition of toxicity would not be possible because of the absence of subjective evidence. Furthermore, neostigmine, which is less toxic and which acts promptly, is thoroughly efficacious. Should the infant have prolonged myasthenic symptoms for as long as a few months after treatment with neostigmine, as in one of the reported cases, experimentation with tetra-ethyl pyrophosphate, shifting gradually from neostigmine therapy, might prove worth while.

NEWS

COMING MEETINGS AND CLINICS

Texas Medical Association, Dallas, May 4-7, 1952. Dr. Allen T. Stewart, Lubbock, Pres.; Tod Bates, 700 Guadalupe St., Austin, Executive Secy.

American Medical Association, Atlantic City, June 11-15, 1951. Dr. Elmer L. Henderson, Louisville, Ky., Pres.; Dr. George F. Lull, 535 North Dearborn St., Chicago 10, Secy.

NATIONAL AND REGIONAL

American Academy of Allergy. Dr. Walter S. Burrage, Milwaukee, Pres.; Mr. James O. Kelley, 208 E. Wisconsin Ave., Milwaukee 2, Executive Secy.

American Academy of Dermatology and Syphilology. Dr. Earl D. Osborne, Buffalo, N. Y., Pres.; Dr. John E. Rauschkolb, 25 Prospect Ave., N. W., Cleveland 15, Secy.

American Academy of General Practice. Stanley R. Truman, Oakland, Calif., Pres.; Mr. Mac F. Cahal, 406 W. 34th St., Kansas City 2, Executive Sec'y.

American Academy of Neurological Surgery. Dr. William S. Keith, Toronto, Canada, Pres.; Dr. T. C. Erickson, 1300 University Ave., Madison 5, Wis., Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 14-18, 1951. Dr. Derrick Vail, Chicago, Pres.; Dr. W. L. Benedict, 100 First Ave. Bldg., Rochester, Minn., Secy.

American Academy of Pediatrics, Toronto, Oct. 20-25, 1951. Dr. Paul W. Beaven, Rochester, N. Y., Pres.; Dr. C. G. Grulee, 636 Church St., Evanston, Ill., Secy.

American Association for Thoracic Surgery, Atlantic City, N. J. Dr. Alfred Blalock, Baltimore, Pres.; Dr. Brian Blades, 901 23rd St. N. W., Washington, D. C., Secy.

American Association of Genito-Urinary Surgeons, Skytop, Pa., May 16-18, 1951. Dr. Roger C. Graves, Boston, Pres.; Dr. Norris J. Heckel, 122 S. Michigan Ave., Chicago 3, Secy.

American Association of Obstetricians, Gynecologists, and Abdominal Surgeons, Hot Springs, Va., Sept. 6-8, 1951. Dr. James K. Quigley, Rochester, N. Y., Pres.; Dr. William F. Mengert, 2211 Oak Lawn Ave., Dallas, Secy.

American Cancer Society. Dr. Guy Aud, Louisville, Ky., Pres.; Mr. M. R. Runyon, 47 Beaver St., New York, Exec. Vice-Pres.

American College of Allergists. Dr. John H. Mitchell, Columbus, Ohio, Pres.; Dr. Fred W. Wittich, 423 La Salle Medical Building, Minneapolis 2, Secy.

American College of Chest Physicians, Atlantic City, N. J., June 7-10, 1951. Dr. Louis Mark, Columbus, Ohio, Pres.; Mr. Murray Kornfeld, 500 N. Dearborn St., Chicago 10, Executive Secy.

American College of Physicians. Dr. William S. Middleton, Madison, Wis., Pres.; Mr. E. R. Loveland, 4200 Pine St., Philadelphia 4, Secy.

American College of Radiology, Atlantic City, June 10, 1951. Dr. C. Edgar Virden, Kansas City, Mo., Pres.; Mr. W. C. Stronach, 20 N. Wacker Drive, Chicago 6, Executive Secy.

American College of Surgeons San Francisco Nov. 5-9, 1951. Dr. Henry W. Cave, New York, Pres.; Dr. Paul B. Magnuson, 40 E. Erie St., Chicago 11, Secy.

American Congress of Physical Medicine, Denver, Sept. 3-8, 1951. Dr. Arthur L. Watkins, Boston, Pres.; Dr. Richard Kovacs, 2 E. 88th St., New York 28, Secy.

American Dermatological Association, Hot Springs, Va., May 23-26, 1951. Dr. Henry E. Michelson, Minneapolis, Pres.; Dr. L. A. Brunsting, 102 2nd Ave., S. W., Rochester, Minn, Secy.

American Gastro-Enterological Association, Atlantic City, June 8-9, 1951. Dr. John G. Mateer, Detroit, Pres.; Dr. Dwight L. Wilbur, 655 Sutter St., San Francisco, Secy.

American Gynecological Society, New York, May 7-9, 1951. Dr. Frederick Irving, Brookline, Pres.; Dr. Norman F. Miller, 1313 E. Ann St., Ann Arbor, Mich., Secy.

American Hospital Association. Dr. Charles F. Wilinsky, Boston, Pres.; Mr. George P. Bugbee, 18 E. Division St., Chicago, Executive Secy.

American Laryngological, Rhinological, and Otological Society, White Sulphur Springs, W. Va., May 6-8, 1951. Dr. Gordon B. New, Rochester, Minn., Pres.; Dr. Louis H. Clerf, 1530 Locust St., Philadelphia, Secy.

American Neurological Association, Atlantic City, June 18-20, 1951. Dr. Wilder Penfield, Montreal, Canada, Pres.; Dr. H. Houston Merritt, 710 W. 168th St., New York 32, Secy.

American Ophthalmological Society, White Sulphur Springs, W. Va., June 7-9, 1951. Dr. John H. Dunnington, New York, Pres.; Dr. M. C. Wheeler, 30 W. 59th St., New York 19, Secy.

American Orthopedic Association, White Sulphur Springs, W. Va., June 20-23, 1951. Dr. James S. Speed, Memphis, Pres.; Dr. C. Leslie Mitchell, Henry Ford Hospital, Detroit 2, Secy.

American Pediatric Society, Atlantic City, May 2-5, 1951. Dr. Bronson Crothers, Boston, Pres.; Dr. Henry G. Poncher, 1819 W. Polk St., Chicago 12, Secy.

American Proctologic Society, Atlantic City, June 7-10, 1951. Dr. Hoyt R. Allen, Little Rock, Pres.; Dr. W. Wendell Green, 1838 Parkwood Ave., Toledo 2, Secy.

American Psychiatric Association, Cincinnati, May 7-11, 1951. Dr. John C. Whitehorn, Baltimore, Pres.; Dr. R. Finley Gayle, 501 E. Franklin St., Richmond, Va., Secy.

American Public Health Association, San Francisco, Oct. 29-Nov. 2, 1951. Dr. W. P. Shepard, San Francisco, Pres.; Dr. R. M. Atwater, 1790 Broadway, New York 19, Secy.

American Society of Anesthesiologists, Washington, D. C., Nov. 5-8, 1951. Dr. Urban H. Eversoll, Boston, Pres.; Dr. J. E. Remlinger, Jr., 188 W. Randolph St., Chicago, Secy.

American Society of Clinical Pathologists, Chicago, Oct. 13-20, 1951. Dr. F. William Sundermann, Atlanta, Ga., Pres.; Dr. Clyde G. Culbertson, Indiana University School of Medicine, Indianapolis, Secy.

American Surgical Association. Dr. Daniel C. Elkin, Atlanta, Pres.; Dr. Nathan Womack, University of Iowa, Iowa City, Secy.

American Urological Association, Chicago, May 21-24, 1951. Dr. Thomas D. Moore, Memphis, Tenn., Pres.; Dr. C. H. deT. Shivers, 121 S. Illinois Ave., Atlantic City, N. J., Secy.

Association of American Physicians and Surgeons, Indianapolis, Oct. 4-6, 1951. Dr. Lawerence Shinabery, Fort Wayne, Ind., Pres.; Mr. Harry E. Northam, 360 N. Michigan Ave., Chicago 1, Executive Secy.

International College of Surgeons, U. S. Chapter, Chicago, Sept. 11-14, 1951. Dr. Custis Lee Hall, Washington, D. C., Pres.; Dr. Arnold S. Jackson, 1516 Lake Shore Drive, Chicago, Secy.

National Tuberculosis Association, Cincinnati, May 14-18, 1951. Dr. David T. Smith, Durham, N. C., Pres.; Dr. James E. Perkins, 1790 Broadway, New York 19, Managing Director.

Radiological Society of North America, Chicago, Dec. 3-7, 1951. Dr. John S. Bouslog, Denver, Pres.; Dr. D. S. Childs, Medical Arts Bldg., Syracuse 2, N. Y., Secy.

Southern Medical Association, Dallas, Nov. 5-8, 1951. Dr. Curtice Rosser, Dallas, Pres.; Mr. C. P. Loranz, 1020 Empire Bldg., Birmingham, Ala., Secy.

Southern Psychiatric Association. Dr. R. Burke Suitt, Durham, N. C., Pres.; Dr. Newdigate M. Owensby, Medical Arts Bldg., Atlanta, Ga., Secy.

Southern Surgical Association, Hot Springs, Va., Dec. 4-6, 1951. Dr. Frank C. Wilson, Birmingham, Ala., Pres.; Dr. John C. Burch, 2112 West End Ave., Nashville, Tenn., Secy.

Southwest Allergy Forum. Dr. J. H. W. Rouse, San Antonio, Pres.; Dr. Boen Swinny, 224 Medical Arts Bldg., San Antonio 5, Secy.

Southwest Regional Cancer Conference. Secy., 209 Medical Arts Bldg., Fort Worth.

Southwestern Medical Association, El Paso, Oct. 18-20, 1951. Dr. L. W. Breck, El Paso, Pres.; Dr. W. W. Schuessler, 1415 First National Bank Bldg., El Paso, Secy.

Southwestern Surgical Congress, St. Louis, Sept. 24-26, 1951. Dr. Leo J. Starry, Oklahoma City, Pres.; Dr. C. R. Rountree, 1227 Classen, Oklahoma City 3, Secy.

Tri-State Medical Assembly. Dr. W. S. Terry, Jefferson, Pres.; Dr. James Harris, Marshall, Secy.

United States-Mexico Border Public Health Association. Dr. Luis Arriaga Velez, Chihuahua, Mexico, Pres.; Dr. M. F. Haralson, 314 U. S. Court House, El Paso, Secy.

STATE

Texas Academy of General Practice, Houston, September 10-11, 1951. Dr. Andrew S. Tomb, Victoria, Pres.; Dr. B. H. Bayer, 104 E. Twentieth St., Houston, Secy.

Texas Academy of Internal Medicine, Galveston, December 8-9, 1951. Dr. W. L. Marr, Galveston, Pres.; Dr. John S. Chapman, 3810 Swiss Ave., Dallas, Secy. Meetings restricted to members.

Texas Air-Medics Association. Dr. D. P. Laugenour, Dallas, Pres.; Dr. C. F. Miller, 906 Medical Arts Bldg, Waco, Secy.

Texas Association of Obstetricians and Gynecologists, Austin, Feb. 8-9, 1952. Dr. S. Foster Moore, San Antonio, Pres.; Dr. Carey Hiett, 603 College Ave., Fort Worth, Secy.

Texas Chapter, American College of Chest Physicians. Dr. David McCullough, Kerrville, Pres.; Dr. Henry R. Hoskins, 514 Medical Arts Bldg., San Antonio, Secy.

Texas Club of Internists. Dr. Ghent Graves, Houston, Pres.; Dr. Hatch W. Cummings, Jr., Houston, Secy.

Texas Dermatological Society. Dr. J. G. Brau, Dallas, Pres.; Dr. W. Harris Connor, 601 Medical Arts Bldg., Houston, Secy.

Texas Diabetes Association. Dr. B. F. Smith, Houston, Pres.; Dr. W. N. Powell, W. Ave. F, Temple, Secy.

Texas Division, American Cancer Society, Fall, 1951. Mr. Frank C. Smith, Houston, Pres.; Mr. J. Louis Neff, 2307 Helena St., Houston 6, Executive Director.

Texas Heart Association. Dr. George W. Parson, Texarkana, Pres.; Miss Roberta Miller, 411 Reserve Loan Life Bldg., Dallas, Executive Secy.

Texas Hospital Association. Mr. Roy Wilmesmeier, Houston, Pres.; Mrs. Ruth Barnhart, 2210 Main St., Dallas, Secy.

Texas Neuropsychiatric Association. Dr. Martin L. Towler, Galveston, Pres.; Dr. James Blair, San Antonio, Secy.

Texas Orthopedic Association. Dr. Louis Breck, El Paso, Pres.; Dr. Margaret Watkins, 3629 Fairmount St., Dallas, Secy.

Texas Pediatric Society, Houston, October 12-13, 1951. Dr. Thomas D. McCrummen, Austin, Pres.; Dr. M. C. Carlisle, 1410 Austin Ave., Waco, Secy.

Texas Public Health Association. Mr. Barnie A. Young, Austin, Pres.; Mr. Earle W. Sudderth, Dallas County Health Department, Court House, Dallas, Executive Secy.

Texas Radiological Society. Curtis H. Burge, Houston, Pres.; Dr. R. P. O'Bannon, 650 Fifth Ave., Fort Worth, Secy.

Texas Railway and Traumatic Surgical Association. Dr. R. J. White, Fort Worth, Pres.; Dr. W. F. Parsons, First National Bank Bldg., Fort Worth, Secy.

Texas Rheumatism Association, Galveston, December, 1951. Dr. G. W. N. Eggers, Galveston, Pres.; Dr. Robert H. Mitchell, 210 Medical Arts Bldg., Fort Worth, Secy.

Texas Society for Mental Health. Dr. Warren T. Brown, Houston, Pres.; Mrs. Elizabeth F. Gardner, 2504 Jarratt Ave., Austin 21, Executive Secy.

Texas Society of Anesthesiologists. Dr. Russell Bonham, Houston, Pres.; Dr. Werner Hoeflich, 2301 Reba, Houston, Secy.

Texas Society of Gastroenterologists and Proctologists. Dr. Carl G. Giesecke, San Antonio, Pres.; Dr. John S. Bagwell, Medical Arts Bldg., Dallas, Secy.

Texas Society of Ophthalmology and Otolaryngology, Austin, Dec. 7-8, 1951. Dr. Edward D. Dumas, San Antonio, Pres.; Dr. Lyle Hooker, 627 Esperson Bldg., Houston, Secy.

Texas Society of Pathologists, Galveston, May 2, 1951. Dr. Stuart A. Wallace, Houston, Pres.; Dr. A. O. Severance, 205 Camden, San Antonio, Secy.

Texas Surgical Society. Dr. Edward White, Dallas, Pres.; Dr. Truman G. Blocker, 927 Strand, Galveston, Secy.

Texas Tuberculosis Association. Dr. Elliott Mendenhall, Dallas, Pres.; Miss Pansy Nichols, 208 E. Ninth, Austin, Executive Secy.

Texas Urological Society, Dallas, 1952. Dr. William H. Heck, San Antonio, Pres.; Dr. J. D. Mitchell, 1414 Medical Arts Bldg., Dallas, Secy.

DISTRICT

Second District Society. Dr. A. H. Daniell, Brownfield, Pres.; Dr. C. B. Knox, Jr., Seagraves, Secy.

Third District Society. Dr. Allen T. Stewart, Lubbock, Pres.; Dr. James T. Hall, 1302 Avenue Q, Lubbock, Secy.

Fourth District Society, Brady, November, 1951. Dr. S. Braswell Locker, Brownwood, Pres.; Dr. B. A. Hallum, Brady, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 6-7, 1951. Dr. E. King Gill, Corpus Christi, Pres.; Dr. Foy Moody, 1611 Fifth St., Corpus Christi, Secy.

Seventh District Society, June, 1951. Dr. John F. Thomas, Austin, Pres.; Dr. George W. Tipton, 502 W. 15, Austin, Secy.

Eighth District Medical Society. Dr. Leonard Johnson, El Campo, Pres.; Dr. Robert Casey, Texas City, Secy.

Ninth District Society. Dr. F. Ray Black, Huntsville, Pres.; Dr. Lyman C. Blair, 1212 Rothwell, Houston, Secy.

Tenth District Medical Society. Dr. T. A. Fears, Beaumont, Pres.; Dr. J. M. White, Jr., 3149 Proctor, Port Arthur, Secy.

Eleventh District Society, Athens. Dr. Griff T. Ross, Mount Enterprise, Pres.; Dr. John M. Travis, Jr., Jacksonville, Secy.

Twelfth District Society, Bryan, July 10, 1951. Dr. S. C. Richardson, Bryan, Pres.; Dr. J. C. Terrell, Stephenville, Secy.

Thirteenth District Society, Fort Worth, June 27, 1951. Dr. R. L. Daily, Wichita Falls, Pres.; Dr. S. W. Wilson, Medical Arts Bldg., Fort Worth, Secy.

Fourteenth District Society, McKinney, June 12, 1951. Dr. Mayo Tenery, Waxahachie, Pres.; Dr. L. W. Johnston, 502 W. College St., Terrell, Secy.

Fifteenth District Society, 1951. Dr. R. G. Granbery, Marshall, Pres.; Dr. H. O. Padgett, Marshall, Secy.

CLINICS

Dallas Southern Clinical Society. Miss Betty Elmer, Medical Arts Bldg., Dallas 1, Executive Secy.

International Post-Graduate Medical Assembly of Southwest Texas, San Antonio, Jan. 22-24, 1952. Dr. John J. Hinchey, P. O. Box 2445, San Antonio, Secy.

New Orleans Graduate Medical Assembly. Dr. Woodard D. Beacham, Room 105, 1430 Tulane Ave., New Orleans 12, Secy.

North Texas-Southern Oklahoma Fall Clinical Conference, Wichita Falls, Sept. 19, 1951. Dr. W. L. Powers, Hamilton Bldg., Wichita Falls, Chairman.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 29-Nov. 1, 1951. Mrs. Muriel R. Waller, 512 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Post Graduate Medical Assembly of South Texas, Houston, July 23-25, 1951. Dr. C. A. Dwyer, Secy., 229 Medical Arts Bldg., Houston.

New Baylor Hospital Blood Center

Construction of a two-story building to house an institute for research on blood diseases was started on Baylor Hospital grounds in Dallas in March. More than $600,000 was donated by Mr. and Mrs. J. K. Wadley of Texarkana to establish the J. K. and Susie L. Wadley Research Institute and Blood Bank as a memorial to their grandson, Keener Bob Moseley, who died of leukemia.

Dr. J. M. Hill, director of the graduate research institute and blood center of Baylor Hospital, will be director of the new blood center. The building is expected to be completed in about one year.

INCREASE IN LIFE EXPECTANCY

A nineteen-year gain in the average length of life in the United States during the past fifty years can be increased further depending upon the reduction or postponement of deaths from chronic conditions, especially the diseases of the circulatory system and cancer. A study on the increase of life expectancy made by Metropolitan Life Insurance Company statisticians reveals that the nineteen-year gain of past years can be attributed to reduction in mortality from the infectious diseases such as pneumonia, influenza, tuberculosis, and the communicable diseases of childhood.

Complete elimination of the heart and blood vessel diseases as a cause of death would increase the expectation of life at birth almost 10 years for men and 9 years for women, while the elimination of cancer would add almost 2 years for men and 2½ years for women.

Course in Radiation Physics

A special course in radiologic physics will be offered by the Department of Physics of the M. D. Anderson Hospital for Cancer Research, June 18 to 23. The course, which is to consist of fifteen lectures addressed to physics graduates, biologists, isotope workers, and others interested in the application of radiation physics to medicine and biology, will be taught by Dr. Leonard G. Grimmett, professor of biophysics at the University of Texas Postgraduate School of Medicine, Houston.

Further information may be obtained from the Office of the Dean, Postgraduate School of Medicine, Hermann Professional Building, Houston 6.

National Muscular Dystrophy Research Foundation

Directors for the National Muscular Dystrophy Research Foundation, Inc., were elected at the first annual meeting held March 12 at Baylor University College of Medicine, Houston.

The directors are Dr. William P. Boger, Philadelphia; Dr. D. Denny-Brown, Dr. Madelaine R. Brown, and Dr. Henry R. Viets, Boston; F. R. C. Brown, Dr. Jack E. Ewalt, Dr. William S. Fields, Mrs. Max Kay, Dr. Robert C. L. Robertson, and Dr. Byron P. York, Houston; J. J. Brown, Austin; Dr. Russell N. DeJong, Ann Arbor, Mich.; Dr. A. L. Delaney, Mr. and Mrs. A. J. Hartel, Jr., Dr. Don P. Schulz, Miss Sallie Woods, and Miss Nadine Woods, Liberty; Dr. H. L. January, Albuquerque, N. Mex.; Dr. S. Morgulis, Omaha, Neb.; and Dr. Frank H. Tyler, Salt Lake City.

Officers of the foundation are Dr. Delaney, president; Mrs. Brown, vice-president; Misses Sallie and Nadine Woods, executive secretaries; and Mr. Hartel, treasurer.

The purposes of the foundation are "to guide, unite, encourage, coordinate, and promote the knowledge of and the work being done on any and all phases of muscular dystrophy, including study and research into the cause, nature, and method of prevention of the disease, and the prevention of the harmful after-effects of the disease."

UNIVERSITY OF TEXAS MEDICAL BRANCH

Specialists from many medical centers participated in the ninth postgraduate tumor conference at the University of Texas School of Medicine held March 7-10 in Galveston. The conference was sponsored by the postgraduate division of the Medical Branch and the State Department of Health. Dr. Paul Brindley, professor of pathology, was chairman of the conference.

Visiting speakers at the conference were Drs. A. C. Broders, consultant in surgical pathology, Mayo Clinic, Rochester, Minn.; Thomas Burford, associate professor of surgery, Washington University, St. Louis; Charles F. Crain, radiologist, Alice; Leonard C. Grimmett, professor of biophysics, University of Texas Postgraduate School of Medicine, Houston; Jarrell E. Miller, clinical associate professor of radiology, Southwestern Medical School, Dallas; Donald S. Morris, director of pathological anatomy, Scott and White Clinic, Temple; James Davidson Rives, professor of surgery, Louisiana State University School of Medicine, New Orleans; and J. B. Trunnell, assistant professor of medicine, University of Texas Postgraduate School of Medicine, Houston; and Louis J. Neff, executive director of the Texas Division, American Cancer Society, Houston.

Recent lecturer at the Medical Branch was Charles H. Townes, Ph. D., of the radiation laboratory of Columbia University, New York. Sponsored by Sigma Xi, honorary scientific society, Dr. Townes presented a series of lectures on microwave spectroscopy.

Dr. George R. Herrmann, professor of medicine, was guest speaker at the Louisiana State Medical Society meeting in New Orleans on May 8 and for the New Orleans Academy of Internal Medicine on May 10.

A fellowship for special work in the Tissue Culture Laboratory has been established by the Ferris and Florence Smith Foundation for Plastic Surgical Research, Grand Rapids, Mich. The fellowship is currently held by Dr. Jorge Gonzalez Ramirez of Mexico City.

Dr. Henry R. Viets, librarian of the Boston Medical Library, neurologist, and chairman of the Council on Scientific Assemblies for the American Medical Association, has been appointed lecturer in neurology and historical and cultural medicine at the Medical Branch. Dr. Viets plans to spend a week each semester in Galveston for lectures and conferences.

PERSONALS

Dr. E. S. McLarty, Galveston, is the newly-appointed health officer for Galveston County, reports the University of Texas Medical Branch Alumni *Bulletin.*

Dr. Cornelius Pugsley, Houston, is the new chief of staff of Hermann Hospital. Other appointments reported in the *Bulletin* of the University of Texas Alumni Association are *Dr. W. A. Senglemann* as president of the medical staff of Memorial Hospital, Houston, and *Dr. E. F. Jones* as president of the staff of St. Mary's Infirmary, Galveston.

A certificate of merit was awarded to *Dr. Charles Phillips,* Temple, at the seventh annual meeting of the clinical conference of the Chicago Medical Society for an exhibit, "Sunlight and the Cancer Skin," prepared with *Dr. E. N. Walsh,* Temple, announces the Temple *Telegram.* The exhibit first received recognition at the American Medical Association meeting in San Francisco last year.

Dr. W. F. Starley, Galveston, has been elected surgeon for the Galveston's Gary Ord Camp No. 7, United Spanish War Veterans, according to the Galveston *News.*

Dr. and Mrs. P. M. Prieto, El Paso; *Dr. and Mrs. P. Jimenez,* Corpus Christi; and *Dr. and Mrs. E. S. Darsey,* Crockett, are the parents of sons born recently. New daughters have arrived for *Dr. and Mrs. T. M. Oliver,* Waco; *Dr.*

and Mrs. J. W. Kopecky, San Antonio; and *Dr. and Mrs. N. F. McDonald,* San Antonio, reports the *Bulletin* of the University of Texas Medical Branch Alumni Association.

Dr. William F. Mengert, professor of obstetrics at the Southwestern Medical School in Dallas, recently described new obstetric techniques at a meeting of the Chicago Medical Society, the Dallas *Times-Herald* states.

Two Texas physicians who observed fifty years of practice recently were *Dr. C. S. Carter,* Bells, and *Dr. Clifton Cheatham,* Wolfe City, report the Dallas *Morning News* and the Ladonia *News.*

Dr. Leo S. Duflot, Galveston, and Miss Rosemary Collins were married recently, states the *Bulletin* of the University of Texas Medical Branch Alumni Association.

LIBRARY SECTION

PACKAGE SERVICE

The package library consists of collections of reprints and other periodical material on various subjects, prepared for lending to members of the Association. Request for packages should be addressed "Library, Texas Medical Association, 700 Guadalupe Street, Austin, Texas." Twenty-five cents in stamps should be enclosed with the request to cover postage and part of the expense of collecting the material. Packages are allowed to remain in the hands of the borrower for 14 days.

ACCESSIONS

The following additions were made to the Library during April:

Reprints received, 1,067.

Journals received, 295.

Books received, 19.

Cancer As I See It, Abelmann, Philosophical Library, Inc., New York.

1950 Yearbook of Neurology, Psychiatry and Neurosurgery, Mackay, editor; *1950 Yearbook on Dermatology and Syphilology,* Baer, Yearbook Publishers, Chicago, Ill.

Surgical Forum, Proceedings Thirty-Sixth Clinical Congress, American College of Surgeons; *Textbook of X-Ray Diagnosis,* Volume II, Shanks; *Diabetes Mellitus, Principles and Treatment,* Duncan; *Electroencephalography, Clinical Practice,* Schwab; *The Neurosis, Diagnosis and Management,* Alvarez, W. B. Saunders Company, Philadelphia & London.

Neurosurgical Treatment of Traumatic Paraplegia, Pool; *Chronic Ulcerative Colitis,* Bargen; *Medical Neuropathology,* Scheinker; *Transactions of the American Goiter Association, 1950,* American Goiter Association; *Fever Therapy,* Kendell; *Blood Groups in Man,* Race and Donger; *Tuberculosis, Among Children and Adults,* Myers; Charles C. Thomas, Springfield, Ill.

Hospital Staff and Office Manual, Larkowski, Romaine Pierson Publishers, Inc., Great Neck, N. Y.

Lung Dust Lesions vs. Tuberculosis, Cole, American Medical Films, Inc., White Plains, N. Y.

Handbook of Pediatric Medical Emergencies, Varga; *Immunology,* Sherwood and Sylzberger, C. V. Mosby Company, St. Louis.

Chronic Illness, Turner, Consultant, Federal Security Agency, Public Health Service, United States Government Printing Office, Washington.

Vitamin E. Annotated Bibliography, 1940-1950, Harris and Kujawski, Distillation Products, Industries, Rochester, N. Y.

Industrial Health and Medical Progress, Klem, McKiever, and Lear, Federal Security Agency, Public Health Service, Division of Industrial Hygiene, Washington, D. C.

SUMMARY OF SERVICE

Local users, 53.	Borrowers by mail, 40.
Local packages, 28.	Packages mailed, 57.
Items borrowed, 378.	Film borrowers, 75.

Films loaned, 65.

MOTION PICTURE FILM LIBRARY

Motion picture films on medical subjects, 16 mm., both silent and sound, some in color, and suitable for either medical or lay audiences, are available for loan to county medical societies, hospital staffs, or individual physicians, on request. Borrowers will be requested to pay only the cost of shipment of the films, by express, with insurance, and for any damage to films in the hands of the borrower.

Request for films should be addressed to "Motion Picture Film Library, Texas Medical Association, 700 Guadalupe Street, Austin, Texas." A list of available films with descriptions, will be furnished on request.

The following films were loaned by the Library during the month of April:

Accent on Use (National Foundation for Infantile Paralysis)—Hendrick Memorial Hospital School of Nursing, Abilene; Brackenridge Hospital School of Nursing, Austin; and Gonzales High School, Gonzales.

Analgesia, Continuous Caudal (Becton, Dickinson and Company)—Drs. Hutchins and Sadler, Conroe.

Anemias, The (Lederle Laboratories)—Dr. M. J. McCallum, Houston.

Anesthesia, Regional (Winthrop Chemical Corporation)—Scott and White Memorial Hospitals School of Nursing, Temple.

Anesthesia with Vinethene (Merck and Company)—Montgomery County Medical Society, Conroe.

Another to Conquer (Texas Tuberculosis Association)—Tyler-Smith County Health Unit, Tyler.

Anoxia, Physiology of (Linde Air Products)—Hendrick Memorial Hospital School of Nursing, Abilene.

Appendicitis in Childhood (Mead Johnson)—Dr. William V. Cruce, Houston.

Appraisal of the Newborn (Mead Johnson)—Nan Travis Hospital, Jacksonville.

As Others See Us (American Hospital Association)—Mrs. Ernest R. Grumbles, Atlanta.

Blood Transfusion (British Information Services)—Wellington P.T.A. and Wellington Schools, Wellington.

Breast Cancer: The Problem of Early Diagnosis (American Cancer Society)—Montgomery County Medical Society, Conroe; Smith County Medical Society, Tyler; and Brackenridge Hospital School of Nursing, Austin.

Breast Self-Examination (American Cancer Society)—Brackenridge Hospital School of Nursing, Austin, and The Johns Clinic and Hospital, Taylor.

Breech Extraction with Forceps (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple, and Orange City Hospital Nursing Staff, Orange.

Cardiovascular Anomalies, Congenital, Amenable to Surgery (Mead Johnson)—Smith County Medical Society, Tyler.

Cervical Smears (Dr. Karl John Karnaky)—University of Texas College of Pharmacy, Austin.

Cesarean Section, Low Cervical (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple.

Child Study: Life History of Mary (New York University Film Library)—Dr. William V. Cruce, Houston.

Coming Home (Texas Tuberculosis Association)—Tahoka Hospital, Tahoka.

D.D.T., The Story of (British Information Services)—Gonzales High School, Gonzales.

Diphtheria and Croup (Lederle Laboratories)—Scott and White Memorial Hospitals School of Nursing, Temple.

Doctor Speaks His Mind, The (American Cancer Society)—Pewitt Lions Club, Omaha.

Dwarfism (Mead Johnson)—Bastrop Clinic, Bastrop.

Dysmenorrhea, Primary (G. D. Searle and Company)—Sewell Hospital, Belton.

Electric Shock Therapy in Depressions (Drs. Blair and Cooper)—The Johns Clinic and Hospital, Taylor, and Dr. William V. Cruce, Houston.

Esophagus, Surgical Treatment for Carcinoma of the Lower End of (Dr. Philip Thorek)—The Johns Clinic and Hospital, Taylor.

Eyes, Your Children's (British Information Services)—Dr. William V. Cruce, Houston.

Feeding the Infant During the First Year (Mead Johnson)—Nan Travis Hospital, Jacksonville.

Forty Billion Enemies (Westinghouse Electric and Manufacturing Company)—Pewitt Lions Club, Omaha.

Golden Glory (Standard Brands, Inc.)—Jacksonville High School, Jacksonville.

Hepatitis, Observations on (Mead Johnson)—Tahoka Hospital, Tahoka.

Human Fertility, Studies in (Ortho-Products, Inc.)—The University of Houston, Houston.

Hypodermic Syringes and Needles: Their Care and Function (Becton, Dickinson and Company)—Tahoka Hospital, Tahoka.

In Daily Battle (National Foundation for Infantile Paralysis)—University of Texas College of Pharmacy, Austin, and Brackenridge Hospital School of Nursing, Austin.

Infantile Paralysis, Your Fight Against (National Foundation for Infantile Paralysis)—Brackenridge Hospital School of Nursing, Austin.

Lease on Life (United States Public Health Service)—The Johns Clinic and Hospital, Taylor.

Life Begins Again (British Information Services)—Gonzales High School, Gonzales.

Magic Bullets (United States Public Health Service)—Jacksonville High School Physical Education Classes, Jacksonville.

Malaria (British Information Services)—Scott and White Memorial Hospitals School of Nursing, Temple.

Management of the Failing Heart (Varick Pharmacal Company)—Dr. William V. Cruce, Houston, and The University of Texas School of Pharmacy, Austin.

Modern Nutrition (E. R. Squibb and Company)—The University of Texas College of Pharmacy, Austin.

On Our Own (National Foundation for Infantile Paralysis)—Brackenridge Hospital School of Nursing, Austin.

Once Upon A Time (Metropolitan Life Insurance Company)—Jacksonville Public Schools, Jacksonville.

Pneumonia (Mead Johnson)—Tahoka Hospital, Tahoka.

Polio—Diagnosis and Management (British Information Services)—Lillie Jolly School of Nursing, Houston.

Poliomyelitis, Sister Kenny Method of Treatment of (Dr. Herbert Hipps)—Lillie Jolly School of Nursing, Houston.

Psychiatry in Action (British Information Services)—Wichita Falls Clinic Hospital, Wichita Falls.

Resuscitation of the Newborn (Mead Johnson)—Orange City Hospital Nursing Staff, Orange.

Roentgen Pelvimetry (Mead Johnson)—Dr. William V. Cruce, Houston.

Scarlet Fever (Lederle Laboratories)—Scott and White Memorial Hospitals School of Nursing, Temple.

Serum, Human, The Preparation of (Mead Johnson)—Scott and White Memorial Hospitals School of Nursing, Temple.

Stitch in Time, A (American Medical Association)—Gonzales High School, Gonzales.

Sulfonamide Therapy (Lederle Laboratories)—Scott and White Memorial Hospitals School of Nursing, Temple.

TB, This Is (Texas Tuberculosis Association)—Brackenridge Hospital School of Nursing, Austin.

Techniques of Injection (Becton, Dickinson and Company)—Orange City Hospital Nursing Staff, Orange.

They Also Serve (American Medical Association)—Baylor University Medical College, Houston.

They Do Come Back (Texas Tuberculosis Association)—Tahoka Hospital, Tahoka, and Brackenridge Hospital School of Nursing, Austin.

Tuberculosis, Role of Public Health Nurse in (Texas Tuberculosis Association)—Brackenridge Hospital School of Nursing, Austin.

Urologic Conditions, Diagnosis of (Winthrop Chemical Company)—Medical and Surgical Clinic, Laredo.

Uterosalpingography (E. Fougera and Company, Inc.)—Orange City Hospital Nursing Staff, Orange.

Varicose Veins and Their Complications (Becton, Dickinson and Company)—Sewell Hospital, Belton.

Varicose Veins, Treatment of (G. D. Searle and Company)—Dr. William V. Cruce, Houston.

Vitamin A in Human Nutrition (Mead Johnson)—The Johns Clinic and Hospital, Taylor.

What Is Cancer? (American Cancer Society)—Bastrop Clinic, Bastrop.

You Can Help (Texas Tuberculosis Association)—Texas Tuberculosis Association, Annual Meeting, Dallas.

BOOK NOTICES

[1]Pediatric X-Ray Diagnosis

John Caffey, A. B., M. D., Professor of Clinical Pediatrics, College of Physicians and Surgeons, Columbia University; Attending Pediatrician and Roentgenologist, Babies Hospital and Vanderbilt Clinic, New York. Second edition. Cloth, 862 pages. $22.50. Chicago, Year Book Publishers, 1950.

This is the second edition of Dr. Caffey's book on pediatric roentgen diagnosis. Several changes have been made in this edition, notably a more detailed discussion of the bone changes of hypervitaminosis and cortical hyperostosis.

This book is unquestionably the most authoritative monograph on pediatric roentgen diagnosis.

[2]Brain Metabolism and Cerebral Disorders

Harold E. Himwich, M. D., Chief, Clinical Research Branch, Medical Division, Army Chemical Center, Md. Cloth, 451 pages. $6. Baltimore, Williams and Wilkins Company, 1951.

This book's title is a brief self-summary, but no information less than the book's contents would do it justice, for it is so packed with detailed scientific reports regarding the biology and pathology of the nervous system as to make for a researcher's neurologic haven. The author has consolidated the work done on brain metabolism, has applied it clinically, and has given direction to future research.

The book is done in two closely interrelated parts. The first part deals with cellular physiology and biochemistry; the second concerns the integrative action of the central nervous

[1]*Clifford Thorne, M. D., Austin.*
[2]*Lee F. Scarborough, M. D., Austin.*

system. This combines to give continuity between basic metabolic understandings and clinical application.

The reader is given clear rights to indexed statements, figures, opinions, and authors. The fifty-two page bibliography speaks for itself.

The book is a "must" for those interested in aspects of neurology and psychiatry.

[3]Psychiatry for the Pediatrician

Hale F. Shirley, M. D., Associate Professor of Pediatrics and Psychiatry, Executive Director of the Child Psychiatry Unit, Stanford University School of Medicine. Cloth, 442 pages. New York, Commonwealth Fund, 1948.

The book is a modern concept of the field written by a doctor experienced in child guidance. Some of his essential topics include basic concepts in child guidance; development and habit training; and factors and problems related with physical, intellectual, emotional, sexual, and environmental phases of development. Methods of investigating behavior problems are discussed as are the treatment of behavior problems, including basic principles, interview, and environmental therapy. Finally, the picture of mental health in a changing world is discussed in regard to some of the things that can be accomplished by the pediatrician and general practitioner.

The author cites interesting examples and cases wherever necessary. He also applies simple lay terms and explains most problems in an understanding manner. He always includes a good list of references and bibliographies both for the physicians and the parents.

[4]Basic Principles of Clinical Electrocardiography

Hans H. Hecht, M. D., Associate Professor of Medicine, University of Utah School of Medicine, Salt Lake City. Lexide, 88 pages. $2. Springfield, Ill., Charles C. Thomas, 1950.

This monograph is designed to lead the reader through the maze of concepts of clinical interpretation on one hand and experimentally established fact on the other. Dr. Hecht has achieved his aim with clarity and fidelity to both views. Whereas this is a slim volume, almost every sentence is of succulent and rich substance, and must be thoroughly masticated to be fully digested and appreciated.

The subject matter is plainly written, easily understandable, with little technical language and clear-cut illustrations. The book is recommended for anyone interested in the subject of heart disease.

In conclusion Dr. Hecht has been able to show the "correlation of certain techniques and to integrate various concepts currently applied to the interpretation of electrocardiograms.... It is postulated that if the divergent viewpoints and approaches rest on sound physiological principles, a common basis must be found for all of them."

[5]The Practice of Sanitation

Edward S. Hopkins, Principal Associate Engineer, Bureau Water Supply, Baltimore, Md.; Lieutenant Colonel, Medical Service Corps (Sanitary Engineering Section), United States Army Reserve; Instructor, McCoy College, Johns Hopkins University; and Francis B. Elder, Engineering Associate, American Public Health Association, Colonel, Medical Service Corps (Sanitary Engineering Section), United States Army Reserve. Cloth, 423 pages. $7.50. Baltimore, Williams and Wilkins Company, 1951.

This book is devoted to the field of environmental sanitation, and should be of particular interest to health officers, public health engineers, and sanitarians. Sixty-four pages of this book are devoted to water supply, while other chapters make reference to the latest knowledge on stream pollution, disposal of sewage, refuse, wastes, swimming pools, ventilation, insect and rodent control, food sanitation, and the principles of disinfection.

It is well illustrated and is a good guide in sanitation practices and procedures.

[6]A Pattern for Hospital Care

Eli Ginzberg, Ph. D., Director, New York State Hospital. Cloth, 368 pages. $4.50. New York, Columbia University Press, 1949.

This is a report of a comprehensive study of the hospitals in New York State made by Columbia University and the Joint Hospital Survey and Planning Commission. The survey started August 1, 1948, and was completed September 30, 1949.

At that time there were 543 hospitals in New York with 160,500 beds; of which 21 hospitals with 14,000 beds belonged to federal agencies. The remaining 146,500 beds were distributed as follows: 70,500 to the state, 23,500 to municipalities, 46,000 to voluntary nonprofit agencies, and 6,500 to private organizations.

There is a general discussion of the various types of hospitals, including psychiatric, tuberculosis, and general nursing homes, and out-patient clinics. One chapter discusses the various types of health insurance. A large part of the book deals with the special problems of New York, but most of them are similar to those experienced by other states. Therefore, I recommend this book for hospital administrators, especially those who are connected with state or city-county hospitals. Also, I believe the recommendations made by this committee should be a great help to the members of the hospital boards, the Legislature, and similar bodies who have to prepare budgets for hospitals.

[7]Clinical Applications of Suggestion and Hypnosis

William T. Heron, M. A., Ph. D., Professor of Psychology, University of Minnesota, Minneapolis. Cloth, 116 pages. $3. Springfield, Ill., Charles C. Thomas, 1950.

This is a handbook of slightly more than a hundred pages which gives a short review of the subject including a chapter on the history of hypnosis.

The author describes his work as a handbook and as such it is a valuable addition to the library of the psychologist and psychiatrist. The author is greatly interested in the use of hypnosis in dentistry, a procedure which, although it might make the dreaded experience less dreadful, would be difficult to carry out practically.

The chapters on technique of hypnosis are concise, and there is little excess wordage throughout the volume.

The chapters on suggestion could profitably be read by every physician.

This is a useful, well written, and worth-while handbook on the subject.

[8]Bronchoesophagology

Chevalier Jackson, M. D., Sc. D., LL. D., F.A.C.S.; and Chevalier L. Jackson, M. D., M. Sc., F.A.C.S., Professor of Bronchoesophagology and Laryngeal Surgery, Temple University, Philadelphia. Cloth, 366 pages. $12.50. Philadelphia, W. B. Saunders, 1950.

This book is the latest and best in the field of bronchoesophagology. All chest conditions that may be benefited by bronchoscopy or esophagoscopy are presented.

The first half of the book is devoted to bronchology.

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[3]E. P. Tottenham, M. D., Brenham.
[4]F. B. Faust, M. D., Littlefield.
[5]George W. Cox, M. D., Austin.
[6]R. C. Rowell, M. D., Austin.
[7]Carl Friedman, M. D., Waco.
[8]Roy T. Lester, M. D., Abilene.

While studying this section, the reader is impressed by the thoroughness, the detailed description, and the completeness of the illustrations. The latter include photo-process reproductions of drawings in color, numerous photographic and roentgenographic reproductions, and expert sketches. The anatomy of the tracheobronchial tree and lungs is detailed although concise. The experiences of the authors at the Chevalier Jackson Bronchoscopic Clinic in removing almost 5,000 foreign bodies has clearly shown that in nearly all cases such accidents are preventable. The methods of diagnosis are followed by a description of the technique of removal of foreign bodies including drawings to illustrate selection of instruments, position of the patient on the operating room table, and all other features of the process. Many other conditions such as pulmonary tuberculosis and bronchial tumors are discussed in a comprehensive manner with emphasis being placed on treatment of these conditions from the standpoint of the bronchoesophagologist. The section "Obstructive Conditions of the Bronchial Tree" is thoroughly complete and even includes the technique of bronchospirography.

The second half of the book concerns esophagology. This is also expertly illustrated with drawings, pictures, and roentgenographic reproductions. The esophagoscopic views of the normal as well as numerous pathologic conditions are presented in color to acquaint the reader with what he could expect to see in the living subject. The diseases and abnormalities of the esophagus are discussed in detail.

The authors have successfully presented this work as a practical textbook and a guide to technique. This book is valuable to students of technique. It also will be welcomed by those competent in this field because of its thoroughness and comprehensive management of all the phases of this specialty. It contains all the important newer developments as well as those which have stood the test of time.

[D]Emergencies in Medical Practice

C. Allan Birch, M. D., F.R.C.P., Editor, Physician, Chase Farm Hospital, Enfield. Second edition. Cloth, 564 pages. $5.50. Baltimore, Williams and Wilkins Company, 1950.

The English authors of "Emergencies in Medical Practice" have written an enjoyable book. Medical emergencies that all physicians see are dealt with particularly in a concise, interesting fashion. The treatments described are complete in all respects. The commercial names of some of the drugs are entirely new since the book was written in England. The book is to be recommended especially to those young doctors just starting in private practice.

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[D]*V. E. Friedewald, M. D., Big Spring.*

ORGANIZATION SECTION

AMERICAN MEDICAL ASSOCIATION

Atlantic City Meeting June 11-15

More than 400 papers covering nineteen scientific sections will be presented at the one hundredth annual meeting of the American Medical Association, June 11-15 in Atlantic City, where the largest attendance in the history of the A.M.A. is expected. The 1951 annual session will be the fourteenth convention of the A.M.A. held in Atlantic City.

General headquarters, Registration Bureau, Scientific Exhibit, Technical Exposition, and Information Bureau will be in Convention Hall. General scientific meetings and the inaugural meeting are scheduled to be held in the Convention Hall Ballroom, and the House of Delegates meeting will take place in the American Room of the Hotel Traymore.

Physicians are urged to send reservation requests immediately to Dr. Robert A. Bradley, A.M.A. Subcommittee on Hotels, 16 Central Pier, Atlantic City.

Only fellows, affiliate, associate, and honorary fellows, and invited guests may take part in the work of the sections. Members may register for attendance at meetings. Fellows of the Scientific Assembly are members who have paid 1950 A.M.A. membership dues and who applied and paid their fellowship dues of $5 for the current year. Fellowship cards which are sent to all fellows after payment of dues should be presented at the registration window. Physicians who have not received cards for 1951 may do so by writing to the American Medical Association, 535 North Dearborn Street, Chicago.

Dr. Elliott Mendenhall and Dr. Robert R. Shaw of Dallas will present a paper on "Recent Trends in the Treatment of Pulmonary Tuberculosis" at the general scientific meeting June 12. Section papers are to be presented by the following Texas physicians: Drs. Clarence S. Livingood, Samchai Nilasena, Rufus A. Stevenson, J. Fred Mullins, C. O. Haug, Truman Blocker, and Arild E. Hansen, Galveston; Alvis E. Greer, Michael E. De Bakey, James A. Greene, Frederick R. Guilford, J. Mathews Robison, Everett L. Goar, Louis S. Smith, and Russell J. Blattner, Houston; J. W. Nixon and J. F. Perry, San Antonio; and Warner K. Noell, Randolph Air Force Base. Other Texans will participate in the discussions which follow presentation of the papers.

The nineteen sections of the Scientific Assembly will be represented in the Scientific Exhibit, which will consist of 290 exhibits selected from 428 applications. Texans who will be represented in the Scientific Exhibit are Drs. Everett R. Veirs, E. D. McKay, A. C. Scott, Jr., Paul M. Ramey, and J. F. McKenney, Jr., of Temple; Col. Robert B. Lewis, Randolph Air Force Base; Drs. Joe E. Rude, C. W. Rowe, and A. W. Harrison, Galveston; and Drs. Michael E. De Bakey and Herbert C. Allen, Jr., Houston.

Texas delegates to the House of Delegates will be Drs. Joseph B. Copeland, San Antonio; Truman C. Terrell, Fort Worth; Britton E. Pickett, Sr., Carrizo Springs; Edward H. Cary, Dallas; Robert B. Homan, Jr., El Paso; Allen T. Stewart, Lubbock; and John K. Glen, Houston. Dr. Everett C. Fox, Dallas, will be the delegate for the Section on Dermatology and Syphilology, and Dr. Charles T. Stone, Galveston, for the Section on Internal Medicine.

More than 355 firms will be represented in the Technical Exposition, in which products, medical books, instruments, special foods, and physical therapy apparatus will be displayed and explained by technical experts.

The American Physicians Art Association will have its annual art exhibit during the convention. Two hundred trophies will be awarded. Physicians in the United States, Canada, and Hawaii desiring to participate in the show may write for information to Dr. F. H. Redewill, 760 Market Street, San Francisco.

The thirty-fifth tournament of the American Medical Golfing Association will be held June 11 at the Seaview Country Club. Information may be obtained from Mr. Bob Elwell, secretary-treasurer, 1420 Monroe Street, Toledo 2, Ohio.

Officials of the State Medical Association of Texas applying to the Secretary of State for renewal of the charter granted by Texas to the Association in 1901. Seated left to right are John Ben Shepperd, Secretary of State, and Dr. William M. Gambrell, Austin, President of the Association when renewal of the charter was requested. Standing left to right are Drs. F. J. L. Blasingame, Wharton; Truman C. Terrell, Fort Worth; Merton M. Minter, San Antonio; and G. V. Brindley, Temple, all members of the Board of Trustees, and Tod Bates, Austin, Executive Secretary of the Association. Dr. E. A. Rowley, Amarillo, the fifth Trustee, was unable to be present.

THE STORY OF THREE CHARTERS

This year is one to be remembered long in the history of the Texas Medical Association. A fifty-year extension of the Association's 1901 charter was granted by the Secretary of the State of Texas effective May 1, 1951.

Though it was not the Association's first charter, the 1901 document was the first to have reached full maturity, thereby making an extension necessary. Two other state charters have been granted the Association. The one issued November 27, 1853, incorporating the Medical Association of Texas, was ignored by the reorganizers in 1869, although the Association operated without any other until 1889. The second charter designated the name of the organization to be Texas State Medical Association but was forfeited ten years later for nonpayment of a state franchise tax of only $10.

The story of these three charters reflects the history and struggle of an organization made possible by a handful of the ancestors of today's men in white.

First Charter—1853

An item in the December 11, 1852, issue of the *Texas Gazette* reported a meeting of some Travis County physicians who had resolved to call a statewide convention of doctors for the purpose of organizing a state association. Notices were mailed and on January 17, 1853, thirty-five Texas physicians arrived in Austin via stagecoach, horseback, and railroad.

The pioneers lost no time in getting down to business. On the first day, a committee of five was appointed to petition the Legislature for a charter and another withdrew to draft a constitution and by-laws, which was accepted with few changes the next afternoon. Dr. Harrison Taylor, Harrison County, who had ridden 300 miles on horseback to attend the meeting, was elected the first of eighty-five presidents which were to follow. The first vice-president was Dr. W. A. Morris of Austin, the only 1853 charter signer who lived to see a charter issued in 1901 for a lasting association envisioned by those early pioneers ninety-eight years ago.

In its constitution and by-laws the new Medical Association of Texas set the second Monday of each November as the permanent annual session date and designated Austin as a permanent location for such sessions. The original charter which was approved by the Fifth Legislature on November 7, 1853, and which was signed by Governor J. W. Henderson on November 28, contained some factors regarding component local organizations comparable to those of today.

The first annual session was held in Austin on November

14, 1853. Attendance was fair considering the age of the Association, but the meeting was shadowed by almost prophetic clouds of the strife and struggle which were to beset the organization for more than a half century. Two charter members were expelled; travel was difficult and distances were far; and, last but far from least, it must be remembered that medical controversies in those days were serious and violent.

However, impressive membership certificates written entirely in Latin were issued to the members, and Dr. George Cupples, San Antonio, was elected the second president of the State Medical Association. A third president was not chosen until sixteen years later.

In his presidential address at this first annual session, Dr. Cupples proudly announced the formation of two county societies as provided for in the charter; the first was the Bexar County Medical Society formed soon after the convention meeting in January, and the other was that of Travis County.

New Constitution—1869

The second "annual" meeting of the Association was held in Houston in 1869. When the original group had met in Austin sixteen years before, there were ominous signs of the impending struggle between the North and South. It is not surprising that the first effort to organize seemed doomed because of this. There was also the fact that both medical and political discussions aroused highly individualistic and explosive temperaments. There were the added trials of inadequate transportation and yellow fever epidemics which scourged different parts of Texas. And it is possible that the expulsion of the two charter members had its share in discouraging regular annual meetings.

As Austin doctors had led the way in 1853, so a group of Houston men, with members of the Washington County Medical Society, took the reins in 1869 to fuse the disintegrated Association. The first paragraph of the newly adopted constitution and by-laws reveals that the Association was to be known as the Texas State Medical Association. However, it operated practically under the original charter until 1889.

During the years since the first convention, several encouraging steps had been taken in local areas. Only two county societies had been formed in 1853. Now, as several other counties and areas had banded together and formed local societies, these were invited to send delegates to the Houston meeting. The delegates contributed much to the success of the meeting. The new constitution and by-laws was signed by twenty-eight names and, as at the original meeting, the American Medical Association Code of Ethics of 1847 was adopted.

Once again a new charter was sought from the Legislature, but years passed before it was issued, as the doctors battled successive legislative bodies. The only apparent revision was the change of location for annual sessions—from Austin to Houston, but the secretary's office remained in Austin and was so noted therein. By the time the second charter was granted, however, this section had been modified to allow a majority vote to decide which city would be the location for the next annual session. No doubt this factor and the scheduling of annual meetings in the spring instead of November contributed to the regularity of successive meetings. The following three annual meetings were held in Houston with an average attendance of twenty-five members. Only seven members arrived in Waco in 1873; but in Dallas, the 1874 meeting boasted an attendance of one hundred! The Dallas meeting was mainly responsible for the amendment on annual session location in the charter.

Publication of the 1869 proceedings for distribution "to intelligent citizens" was advocated at the second session. At the third annual session, the secretary proudly announced that 5,000 copies of the proceedings had been made at the cost of only $100. In line with that cost level, three hotels in the host city of Houston that year offered board and lodging to visiting doctors for $2 per day.

Long-winded orations were the style of the era. Minutes of the 1870 meeting reveal a motion duly seconded and carried which reads "... only five minutes allowed each member to speak." Another important step at this meeting was the establishment of a "Slate of Business Procedure." In two annual sessions, the infant organization was making definite plans toward fulfillment of a dream for a professional organization. Each successive meeting reflected its growing up—realization of problems not common to other professions or organizations. Other sessions showed that enthusiasm often overreached the practical for the current moment, but many years later some of those early dreams were brought up again at more propitious times.

At the third annual session, publication of a medical journal was first advocated. This, however, was impossible not only because of meager funds, but also because the failure to obtain a charter rendered the Association unable to conduct business as a corporate body. Two years later, the Association resolved to cooperate with "any professional brother" who could supply such a journal.

Formal scientific papers were read for the first time at the 1871 meeting in Houston. A year later Dr. R. H. Jones, secretary, again reported failure on the charter petition to the Legislature with a few choice words of his own relating to political blackmail. Nevertheless, the charter committee resumed its efforts—seemingly to no avail. The lack of a charter continued to hamper the Association's efforts in many directions.

When the Association met in Dallas in 1874, three far-reaching decisions were made. A library was advocated and begun with the books on hand, and for the first time newspaper reporters and editors were invited to the meetings. Because of the large attendance and yearly increasing number of papers presented, the session days were increased by one to make a four-day meeting.

By 1877, the Association had 262 members and each successive year showed an increase of names on the membership records.

The profession today is concerned over the spread of socialistic influence in medicine and may feel that back in the "good old days" the doctors had no worry on that score. Most of them did not. However, some of the present generation may be surprised to learn that the speech of Dr. R. H. Harrison, Columbus, at the 1880 meeting in Brenham contained the following proposed resolution: "... That this Association looks with grave apprehensions, of the disastrous consequences of our section of the country, upon any extension of the powers of the National Board of Health...." The resolution was tabled. During the heated discussion which followed, the far-sighted pioneer, a past-president, in turn withdrew from the Association; was voted an honorary member; and consented to return to membership. All this happened during one afternoon session.

At the thirteenth annual session, the Association adopted the *Texas Medical and Surgical Record,* published at Galveston, as the official organ of the Texas State Medical Association. The agreement lasted only two years. At the 1882 session, individual members and the *Record* editors waged a violent and bitter feud, orally and in print, over the diagnosis and treatment of one medical case. The outcome was

inevitable. Soon after the Association withdrew from its agreement with the *Record,* it ceased publication.

Organization of a life insurance department, which had been approved at the twelfth meeting, was tabled because the Association still had not been re-incorporated under a new state charter. A resolution for changes in the Medical Practice Act providing for the creation of a State Board of Health and the incorporation of the Association was dismissed as "not necessary to procure a charter." The year was 1884 and the Association still was trying to secure a charter, but an unfriendly Legislature managed to prevent its issue.

The Association was beginning to walk instead of toddle. An encouraging number of 350 physicians of the total 449 members were present at the second meeting held in Dallas in 1886, and for the first time printed programs of the session were distributed.

As more physicians joined the Association, more individual tempers clashed. However, the Association now had become a working unit dealing with its personal and statewide problems with a firmer hand. Already there was evidence of "cleaning house" as is shown by the minutes of the 1887 meeting in Austin. Two members were expelled and another was suspended. Because of the suspended member, a friend brought heated charges against a former president which were withdrawn after the friend cooled off. However, before the meeting was over, three more members wre recommended for expulsion and the charges were turned over to the proper committee for investigation. The secretary reported that the library now had 537 books, and he was directed to secure a room for their storage in the new capitol building. He reported failure the next year, but the Association finally rented adequate quarters elsewhere.

For years the Association had been endeavoring to get the Legislature to pass favorable laws regulating and identifying the various professions in fields related to medicine. The Legislature of 1888 passed a bill regulating dentistry and pharmacy, and upon the announcement of this new bill at the annual session, the members rose in applause. The president was encouraged anew to apply again for a charter and instructed the committee to proceed. The meeting ended on another happy note of information—twenty-four county medical societies were now carried on the secretary's roll.

Second Charter—1889

The next year, 1889, was a big one for the adolescent Association. The charter signed April 23, 1889, in San Antonio by Drs. R. M. Swearingen, F. E. Daniel, T. J. Bennett, and J. Larendon, was ratified by the State Legislature, and the Association was incorporated.

The new charter contained few changes from the 1853 charter. It estimated value of assets at $50; the 1853 charter had stipulated the right to possess property not to exceed the sum of $100,000. The new document also provided for a nine-member Board of Trustees which would be the governing body of the Association.

With the granting of the second charter, the Association should have gained new enthusiasm, purpose, and solidarity. The establishment of a medical department at the University of Texas should have been heartening. However, the general personal discord indicated in the late 1880's continued. Although the number of members increased according to the secretary's roster, the percentage of delinquent members finally resulted in an investigation of local society activities ordered by the president in 1893.

When Dr. C. M. Rosser, vice-president of the Association, made a report of his investigation at the 1894 meeting, it revealed that of twenty-nine county medical societies in existence in Texas, only thirteen were alert and active. This apparent apathy on the part of the members and the nationwide, heated debate over some radical changes in the 1848 Code of Ethics proposed by the American Medical Association, paved the way toward reorganization several years later.

The Texas State Medical Association voted in 1894 to retain the 1848 Code of Ethics along with twenty other state associations, and resolved condemnation of acceptance by *The Journal of the American Medical Association* of advertising of patent and proprietary medicines. The *JAMA* editor was instructed the next year to refuse advertisements for medicines without accompanying formulas, but it was ten years before advertisers were required to adhere strictly to this rule or suffer exclusion of such advertisements.

Some extent of the unrest and disruption of the Association is reflected in the title of an editorial by Dr. F. E. Daniel, editor-publisher of the *Texas Medical Journal,* which aired the problems of the Association. This is particularly apparent when it is realized that the editorial, "Shall We Abolish the State Medical Association?" was written by Dr. Daniel, who had served as secretary of the Association and chairman of publications, and who would serve later as president during the year when the Association definitely decided to establish its own medical journal.

One hundred and fifty physicians registered for the 1895 session in Dallas. The business session was suspended long enough for a special memorial service honoring Dr. George Cupples of San Antonio, who died four days before the session convened. Dr. Cupples had served as president in 1854 and again in 1878, and signed the 1853 charter.

It is not surprising that forfeiture of the 1889 charter was due to carelessness—failure to pay a $10 state franchise tax in 1899. The sincerely concerned pioneers reluctantly recognized that the Texas State Medical Association was rapidly approaching stagnation. Less than one hundred doctors attended the 1896 opening session; only twenty-seven were present to vote on important matters brought up later in the session. Membership continued to fall until it was difficult to count three hundred in 1903 but a total of 2,415 in 1904 reflected the struggle which a few had exerted to keep the organization alive. The turn of the century had seemed to renew the interest of Texas physicians as a whole. Perhaps the courage of the people of Galveston was contagious. They insisted on entertaining the Association in spite of the disastrous flood of 1900 which swept the city. Whatever the cause, the Association was on its way to complete and lasting reorganization in 1901.

Third Charter—1901

After years of wrangling, the American Medical Association's efforts to make county societies the basic unit of both state and national organizations succeeded. It was under this plan that the State Medical Association of Texas was reborn. To obtain a new charter, a change in name was required according to the secretary of the Association. Despite some arguments that a renewal was not necessary, a new charter was issued by the State of Texas on May 25, 1901.

Under this 1901 document the organization came to fruition. Every county in the state became a part of a county medical society and a component of the State Association. Internal differences of opinion were composed within the group, and a sufficiently united front was achieved to make the medical profession of Texas a force of recognized strength. The transition from one charter to another this year is marked not with the vacillation and travail which accompanied the establishment of those earlier charters but rather with a continuing sense of harmony, unity, and purpose, plus a progressive program to which more than 6,000 members contribute.

COUNTY SOCIETIES

Angelina County Society

March 12, 1951

(Reported by Bernard Hyman, Secretary)

Arterial Transfusion—Joe R. Gandy, Houston.
Intramedullary Fixation of Fractures of Femur—Denman C. Hucherson, Houston.
High Lights of Anesthesia—L. F. Schumacher, Jr., Houston.

The above scientific program was presented at the March 12 meeting of the Angelina County Medical Society held at the Lufkin Country Club. Twenty members were present.

Bell County Society

April 4, 1951

(Reported by E. R. Veirs, Secretary)

Differential Diagnosis of Jaundice—Richard D. Haines.
Preparation for Atomic Disaster (motion pictures).

Hanes Hanby Brindley and Robert John Carabasi, Temple, were approved for membership in the Bell County Medical Society at a meeting in Temple on April 4.

Approval was given by the society for the establishment of a regional blood bank in Waco under the auspices of the American National Red Cross. Bell County is to be one of the group serviced by the bank.

The annual session of the State Medical Association was discussed and the above scientific program presented. Fred M. Hammond had charge of the motion picture.

Brazoria County Society

February 23, 1951

Motion pictures on atomic warfare and cancer were shown to members of the Brazoria County Medical Society at a meeting in West Columbia on February 23. The business meeting was preceded by a dinner of the society and the Woman's Auxiliary to the Brazoria County Medical Society.

Brazos-Robertson Counties Society

March 20, 1951

Treatment of Skin Cancer—Dr. James Mitchell Brown, Marlin.

The above scientific program was presented at the Brazos-Robertson Counties Medical Society meeting March 20 in Hearne. Mr. Wallace Dreyer of Marlin assisted Dr. Brown by presenting color slides illustrating the talk.

Clay-Montague-Wise Counties Society

Harold Shilling, Bridgeport, was elected president of the Clay-Montague-Wise Counties Medical Society at a recent meeting in Bowie. David Bryant, Bridgeport, was elected secretary.

Dallas County Society

March 13, 1951

(Reported by W. W. Fowler, Secretary)

Symposium: Influence of Pregnancy on Certain Surgical Situations.
Appendicitis in Pregnancy—Sam Burnett, Dallas.
Carcinoma of Breast in Pregnancy—J. Harold Cheek, Dallas.
Peripheral Vascular Disease in Pregnancy—Dale Austin, Dallas.
Thrombocytopenic Purpura in Pregnancy—R. B. Hunter, Dallas.
Pregnancy and Gallstones—Robert S. Sparkman, Dallas.
Discussion—F. J. Sebastian, Dallas.
Medical Economic News—Mr. Millard Heath, Dallas.

The Dallas County Medical Society met with the medical staff of Baylor University Hospital, Dallas, March 13 after attending a dinner given by Baylor Hospital. Robert F. Short, president of the Baylor medical staff, introduced the scientific program given above. E. R. Hayes, chairman of the Dallas Health Museum Fund Solicitation Committee, spoke briefly requesting contributions.

Physicians elected to membership were Fred W. Bone, Guy K. Driggs, Gwendolyn Crass, George E. Emmett, Alvis F. Johnson, Jr., James C. McNamara, Jr., Edward R. Richardson, and L. R. Turbeville. The new members were introduced by Charles L. Martin, president. S. M. Freedman was nominated for honorary membership in the State Medical Association.

On the recommendation of the board of directors the following physicians were elected to military membership: Ernest M. Bralley, Jr., Jack F. Burnett, Robert G. Campbell, Leonard J. Collins, Allan C. Evans, James E. Harkins, George W. Harwood, W. M. Irving, Jr., Paul E. Klinger, C. James Krafft, Alan E. Lindsay, Walter L. Lumpkin, Ralph D. Mahon, Samuel D. Salem, Robert E. Murphy, Eugene V. Powell, Jr., Frederick J. Krueger, George J. Race, Frank O. Seay, Herbert L. Shessel, Robert W. Talley, W. D. Tigertt, Robert Webb, John F. Wetegrove, Lawrence P. Kleuser, Howard A. Bailey, Maurice L. Fergeson, and W. L. Jack Edwards.

April 10, 1951

(Reported by W. W. Fowler, Secretary)

Uses of Newer Drugs in Practice—Andres Goth, Dallas.
Discussion—Martin Buehler, Dallas, Moderator.
Medical Economic News—Mr. Millard Heath, Dallas.

The above scientific program was presented at the April 10 meeting of the Dallas County Medical Society in the Baylor University Hospital Auditorium, Dallas.

Arch McNeill, chairman of the legislative committee, reported on activities of his committee. Applicants elected to membership in the society were Louis Henry Porter, II, and Richard Edward Block.

Resolutions regarding the death of Dr. Morton L. Mazer and Dr. Guy L. Hacker were adopted.

Eastland-Callahan-Stephens-Shackelford-Throckmorton Counties Society

March 20, 1951

(Reported by W. P. Watkins, Secretary)

Thyroid: Emphasis on Carcinoma—Sol Estes, Abilene.
Acute Influenza—R. L. Holmes, Breckenridge.

Physicians of Callahan County were hosts at the Eastland-Callahan-Stephens-Shackelford-Throckmorton Counties Medical Society meeting March 20 in Cisco.

At the business meeting the society voted to invite wives of the society members to attend the meetings and voted to assist the local cancer crusade.

Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Society

March 20, 1951

Myocardial Infarction Due to Coronary Thrombosis—W. T. McCollum, Oklahoma City.
Discussion—Malcolm Wyatt, Pampa.

Dr. McCollum, internist, spoke on the above subject at the Gray-Wheeler-Hansford-Hemphill-Lipscomb-Roberts-Ochiltree-Hutchinson-Carson Counties Medical Society meeting March 20 at Highand General Hospital, Pampa.

Accomplishments of the Council on Medical Defense of the State Medical Association for the past three years were discussed.

Hale-Floyd-Briscoe-Swisher Counties Society

March 20, 1951

Pyelities of Pregnancy—Jan Werner, Amarillo.

Twenty-four members were present for the March 20 meeting of the Hale-Floyd-Briscoe-Swisher Counties Medical Society in Tulia when the above scientific program was presented. Houston M. Burk, Tulia, introduced the speaker.

Kerr-Kendall-Gillespie-Bandera Counties Society

March 12, 1951

Chest Surgery—David Shefts, San Antonio.

In addition to the above scientific program, a discussion on the Blue Cross Hospital Plan was presented at the meeting of the Kerr-Kendall-Gillespie-Bandera Counties Medical Society and Auxiliary March 12 in Kerrville.

Lamar County Society

March 1, 1951

(Reported by Thomas E. Hunt, Jr., Secretary)

At the March meeting of the Lamar County Medical Society held in Paris, J. A. Stephens and J. L. Jopling, both of Paris, reported on a recent meeting which they attended at the University of Oklahoma Hospital. The doctors presented a scientific program on electrocardiogram interpretations.

April 5, 1951

Fourteen members were present for the dinner and business meeting of the Lamar County Medical Society held April 5 at Paris. O. W. Robinson presided at the meeting.

Potter County Society

March 12, 1951

Psychologic Aspects of Infant Care—J. D. Donaldson, Lubbock.

J. D. Donaldson, Lubbock, was guest speaker for Potter County Medical Society at a meeting March 12 in Amarillo. The society adopted a resolution commending the city for deciding to hire a sanitation expert.

Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Society

March 15, 1951

Urologic Problems in Pediatrics—Malcolm C. Maley, Pecos.

The above scientific program was presented at the dinner meeting of the Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Medical Society held March 15 in Monahans. The Woman's Auxiliary to the county medical society met with the society.

Tarrant County Society

March 20, 1951

(Reported by W. P. Higgins, Jr., Secretary)

W. P. Anthony, Jr., and Charles H. Riddle were elected to membership at the March 20 meeting of the Tarrant County Medical Society held at the United States Public Health Service Hospital, Fort Worth.

J. D. Covert and Lily Roberts were recommended for honorary membership in the State Medical Association. Tribute was paid to the late Dr. John T. Moore of Houston.

It was recommended by the public relations and legislative committee and approved by the members present that the public relations committee and the woman's auxiliary have pages in the *Bulletin* of the Tarrant County Medical Society, that further work and publicity be given to the twenty-four hour call system, that publicity on the adjudication committee be endorsed, and that the speech given before the society by the Hon. Martin Dies be printed in the *Bulletin*. It was also recommended that booklets, "Facts You Should Know About Health Cures," be obtained for distribution, that legislators and business men be honored at the public relations meeting in June, and that the possibility of subscribing to the Public Health Legislative Service be investigated.

Mal Rumph outlined activities of the woman's auxiliary. Changes in regard to the care and control of contagious diseases which were suggested by C. S. E. Touzel, chairman of the public health advisory committee, were accepted by the society, and a resolution favoring support of the April crusade of the American Cancer Society, Texas Division, was adopted.

R. G. Baker announced the meeting of the Thirteenth District Medical Society in Fort Worth on June 27 and discussed the payment of American Medical Association dues.

April 3, 1951

(Reported by W. P. Higgins, Secretary)

Reconstructive Procedures about the Head—John B. Patterson, Fort Worth.

Discussion—J. F. Campbell, William M. Crawford, and Joseph Carpentieri, Fort Worth.

A color slide demonstration of reconstruction procedures about the head was shown at a meeting of the Tarrant County Medical Society in Fort Worth on April 3.

Tribute was paid to Dr. E. M. Sager, who died March 19.

A letter from the Home Service Department of the American Red Cross regarding emergency leaves for service men because of illness in their family was read by Ed Etier. M. C. McCarroll spoke regarding S. B. 1140 to establish and consolidate certain hospital, medical, and public health functions of the government in a Department of Health; S. 337, federal aid to medical education; and S. 445, providing federal aid to medical education; and S. 445, providing

Tom Green-Eight County Society

April 2, 1951

(Reported by H. M. Anderson)

Mediastinal Borders—J. E. Miller, Dallas.

Discussion—Jerome Smith, W. D. Anderson, F. T. McIntire, and F. M. Spencer, San Angelo.

A discussion of the present postgraduate training program at Shannon West Texas Memorial Hospital, San Angelo, was held at the April 2 meeting of the Tom Green-Eight County Medical Society in San Angelo.

A publicity committee was appointed to handle publicity about doctors in the society who attend postgraduate meetings and other matters of interest to the press. Members of the committee are Lloyd R. Hershberger, Perry J. C. Byars, Jr., and Gordon F. Madding.

A letter from the San Angelo Colts was read informing the physicians that numbers could be assigned to each doctor for call purposes at Guinn Field. The secretary was instructed to send a list of the members' names to the San Angelo Colts with a request that such numbers be assigned.

Dr. Miller presented the above scientific paper, illustrating it with roentgenograms.

Wharton-Jackson-Matagorda-Fort Bend Counties Society

March 13, 1951

Degenerative Diseases of the Liver—Charles Stone, Galveston.

At the March 13 meeting of the Wharton-Jackson-Matagorda-Fort Bend Counties Medical Society held in Palacios the above scientific program was presented.

Wichita County Society

March 13, 1951

Subarachnoid Hemorrhage (lantern slides)—Albert D'Errico, Dallas.

Albert D'Errico, Dallas neurosurgeon, spoke to sixty members of the Wichita County Medical Society at a dinner meeting March 13 in the Wichita Falls Country Club. Dr. D'Errico spoke on subarachnoid hemorrhage and displayed slides. R. L. Daily introduced the speaker.

At the business meeting J. A. Little, president, appointed an advisory medical committee to serve under the Wichita County civilian defense program. The committee is composed of William Rosenblatt, chairman, H. D. Prichard, and Steve Powers, all of Wichita Falls; P. E. Fish, Electra; J. L. Steele, Iowa Park; and A. G. Steele, Burkburnett.

AUXILIARY SECTION

NATIONAL CONVENTION

Haddon Hall in Atlantic City will be the scene of the meetings and some of the social events of the Woman's Auxiliary to the American Medical Association, which will meet simultaneously with the A.M.A. June 10-15.

Registration will begin Sunday, June 10, at noon. Committees on nominations, resolutions, revisions, and finance will meet Sunday afternoon. A meeting of the board of directors and a luncheon has been planned for Monday, June 11. Mrs. Arthur A. Herold, Shreveport, La., and Mrs. Harold F. Wahlquist, Minneapolis, President and President-Elect of the Auxiliary, will be honored at a tea June 11 followed by a fashion show that evening in the Convention Hall Ballroom.

Formal opening of the twenty-eighth annual meeting of the Auxiliary will take place Tuesday. Past presidents are to be the honor guests at a luncheon with Mr. Ed Lipscomb, director of Public Relations, National Cotton Council, Memphis, as speaker. Members of the Auxiliary also are invited to attend the opening meeting of the A.M.A. in the Convention Hall Ballroom on Tuesday evening.

General sessions will be held Wednesday and Thursday morning and a luncheon in honor of Mrs. Herold and Mrs. Wahlquist is planned for Wednesday noon. The annual dinner of the Woman's Auxiliary to the A.M.A. for members, husbands, and guests will be held Thursday, June 14, followed by the reception and ball in honor of the President of the A.M.A. at the Hotel Traymore.

Among the officers and committee chairmen of the Woman's Auxiliary to the American Medical Association are two Texans, Mrs. George Turner, El Paso, treasurer; and Mrs. Scott Applewhite, chairman of the finance committee. Texas will also be represented at convention by Mrs. O. W. Robinson, President of the Woman's Auxiliary to the Texas Medical Association.

Information on the annual session may be obtained from Auxiliary headquarters, 535 Dearborn Street, Chicago.

AUXILIARY NEWS

Brazos-Robertson Counties Auxiliary

Officers of the Brazos-Robertson Counties Auxiliary include Mesdames R. M. Searcy, Bryan, president; A. G. McGill, Bryan, vice-president; E. M. Boyd, Hearne, recording secretary; Joseph Cox, Bryan, corresponding secretary; T. T. Walton, Bryan, treasurer; J. P. Fleming, Hearne, and David Brown, Bryan, publicity chairmen; and Roy Smith, Bryan, historian.

The officers were elected March 15 at a business meeting following a tea and book review presented by Mrs. A. T. Dyal. Hostesses were Mesdames Searcy, D. W. Andres, E. H. Kirk, and J. W. Geppert.—Mrs. J. W. Geppert, reporter.

Officers of the Woman's Auxiliary to the Texas Medical Association: President, Mrs. O. W. Robinson, Paris; President-Elect, Mrs. Robert F. Thompson, El Paso; First Vice-President (Organization), Mrs. V. M. Longmire, Temple; Second Vice-President (Physical Examinations), Mrs. John H. Wootters, Houston; Third Vice-President (Today's Health), Mrs. R. T. Travis, Jacksonville; Fourth Vice-President (Programs), Mrs. Cecil O. Patterson, Dallas; Recording Secretary, Mrs. E. W. Coyle, San Antonio; Treasurer, Mrs. John D. Gleckler, Denison; Corresponding Secretary, Mrs. Clarence E. Gilmore, Paris; Publicity Secretary, Mrs. A. H. Neighbors, Sr., Austin; Parliamentarian, Mrs. T. Herbert Thomason, Fort Worth.

Dallas County Auxiliary

Mrs. Ridings E. Lee, Dallas, was elected president of the Woman's Auxiliary to the Dallas County Medical Society, March 7 in Dallas. Other officers named are Mesdames Speight Jenkins, president-elect; Robert Sparkman, first vice-president; Wallace B. Wilkinson, second vice-president; Robert Winn, third vice-president; O. M. Marchman, Jr., recording secretary; A. O. Loiselle, corresponding secretary; W. Leslie Bush, treasurer; Alvin Baldwin, assistant treasurer; H. Leslie Moore, parliamentarian; Truett James, publicity; and U. P. Hackney, historian.

Mrs. Harold Block introduced the guest speaker, Mr. Ben Read III, decorator, who spoke on "Fabrics and Their Relationship to Decorating."—Mrs. Charles D. Bussey, Publicity Chairman.

Navarro County Auxiliary

J. Wilson David, Councilor for the Twelfth District, was host at a luncheon in Corsicana on February 16 complimenting wives of members of the Navarro County Medical Society. At that time an Auxiliary to the Navarro County Medical Society was formed with Mrs. C. L. Gary, Jr., being named chairman. Other officers chosen at the meeting were Mrs. O. C. Bowmer, vice-president, and Mrs. S. H. Burnett, secretary-treasurer.

Mrs. William M. Gambrell, Austin, President of the Woman's Auxiliary to the State Medical Association, spoke on the duties of a doctor's wife and on the threat of government control of medicine. Mrs. Gambrell urged the women to acquaint themselves with legislative affairs.

Mrs. Joseph H. McCracken, Dallas, first vice-president of the Woman's Auxiliary, outlined steps to be taken in organizing an auxiliary.

Organization of the Navarro County Auxiliary was completed March 9 at a meeting in the home of Mrs. W. W. Carter, Corsicana. Mrs. Gary was elected president, Mrs. Paul H. Mitchell, corresponding secretary, and Mrs. Dan Hamill was appointed legislative chairman.—Mrs. C. L. Gary, Jr.

Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Auxiliary

The Woman's Auxiliary to the Reeves-Ward-Winkler-Loving-Culberson-Hudspeth Counties Medical Society met March 15 in Monahans with members of the society for a dinner. After dinner separate business meetings were held with the Auxiliary meeting in the home of Mrs. Fred Applegate.

Mrs. Harold Lindley, Pecos, outgoing president, installed the new officers. These are Mrs. David E. Sauer, Kermit, president; Mrs. W. H. McClure, Kermit, treasurer; and Mrs. W. G. Plinke, Wink, secretary.

Mrs. Sauer spoke on public relations.

Victoria-Calhoun-Goliad Counties Auxiliary

Members of the Victoria-Calhoun-Goliad Counties Auxiliary celebrated Doctor's Day with a breakfast in Victoria on March 7. Mrs. Roy S. Lander and Mrs. Ern C. Mooney, both of Victoria, were in charge of the program.

John Vale, Houston, spoke on "Power of Choice." Red carnations were sent to each practicing physician and surgeon in the three counties on that day.

DEATHS

D. O. JETER

Dr. Drayton O. Jeter, Alpine, Texas, died at his home January 31, 1951, from pulmonary fibrosis and cor pulmonale.

Dr. Jeter was born at Wills Point in 1879 and is the brother of four other physicians. He attended the schools of Van Zandt County and later studied at Baylor University, Waco. In 1902 and 1903, Dr. Jeter attended the old University of Dallas Medical Department and then finished his medical education at the Southwestern University Medical College, Dallas. He first began practicing at Kaufman, but later moved to Murchison. In 1928 Dr. Jeter moved to Alpine and had an active practice there until a few months before his death. In 1948 he and his associate, Dr. Malone V. Hill, purchased the Fuller Building in Alpine and remodeled it, forming the Alpine Clinic and Clinic-Hospital shortly thereafter.

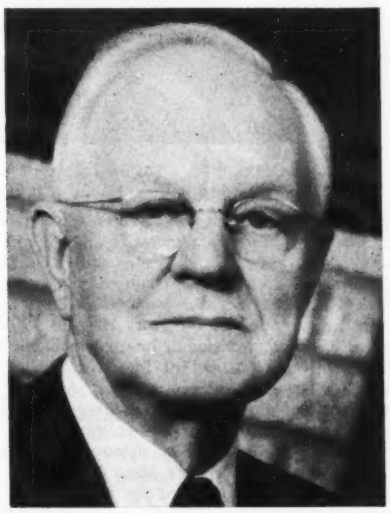
Dr. Jeter was a member of the State Medical Association and the American Medical Association through the Pecos-Jeff Davis-Presidio-Brewster Counties Medical Society, of which he was twice president and once secretary.

He was married to Miss Victoria Kirk of Hickman, Ky., in May, 1908. Besides his wife, an adopted daughter and two sisters survive.

R. E. NICHOLSON

Dr. Richard Eugene Nicholson, Brenham, Texas, died in a Brenham hospital February 7, 1951, of cerebral hemorrhage.

Dr. Nicholson was born September 16, 1874, at Chapel Hill, the son of James Monroe and Lila Margaret (Felder) Nicholson. Receiving his early education from a private tutor and in business college in Austin, he later studied at Southwestern University, Georgetown, and the University of Texas School of Medicine, Galveston. He was graduated from the Medical Department of Tulane University of Louisiana, New Orleans, in 1897, and interned at Chicago Polyclinic Medical College, Chicago. He served in the United States Army during the Spanish-American War and was assistant surgeon with the United States Volunteers Service during World War I. Dr. Nicholson practiced in Chapel Hill before moving to Brenham, where he practiced from 1904 until 1944, when he retired. His specialty was diseases of the eye, ear, nose, and throat.



DR. R. E. NICHOLSON

A member of the American Medical Association, Dr. Nicholson had been a member of the State Medical Association through Washington County Medical Society since 1906. He was elected to honorary membership in the state organization in 1949. In 1934 Dr. Nicholson was elected to fellowship in the American College of Surgeons. Chairman of the board of directors of Farmers National Bank in Brenham, Dr. Nicholson was an honorary steward of the Methodist Church.

On November 24, 1904, in Brenham he married Miss Mary Stone, who preceded him in death May 29, 1931. Survivors are a brother, R. F. Nicholson, Dallas, and a sister, Mrs. Emma Nicholson Buchanan, Brenham.

J. L. ANDERSON

Dr. James LeRoy Anderson, San Antonio, Texas, died February 3, 1951, from coronary thrombosis.

Dr. Anderson was born September 17, 1886, in Ailsa Craig, Ontario, Canada, the son of Dr. James Ross and Katherine Mae (Kay) Anderson. He attended Park Hill High School, Park Hill, Canada, and was graduated from the Western University Faculty of Medicine, London, Ontario, in 1909, after which he interned at Pittsburgh Tuberculosis League Hospital. He was superintendent at Chicago Winfield Tuberculosis Sanatorium, Winfield, Ill., from 1914 until 1918. Dr. Anderson practiced at Minot, N. D., and

DR. J. L. ANDERSON

served in the United States Army Medical Corps. He had been medical director of Grace Lutheran Sanatorium, San Antonio, since 1921. Dr. Anderson was a member of the American College of Chest Physicians and specialized in pulmonary diseases.

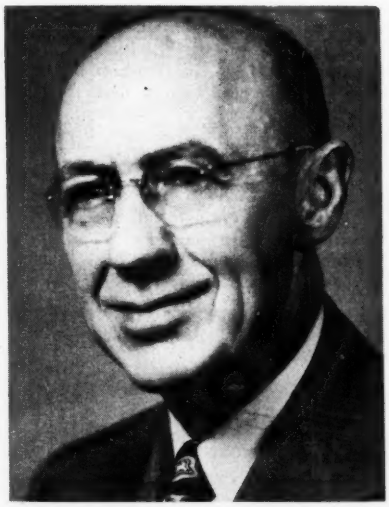
A member of the American Medical Association, he was a member of the State Medical Association through Bexar County Medical Society. He was a member of the Presbyterian Church.

Dr. Anderson is survived by his wife, the former Miss Clare Leland, whom he married in Chicago, October 12, 1915. He is also survived by a sister, Miss Belle Anderson, Toronto, Canada.

T. E. WINFORD

Dr. Thomas Edgar Winford, Dallas, Texas, died at his home March 7, 1951, from adenocarcinoma of the sigmoid.

Born in Lynn, Ark., January 7, 1892, Dr. Winford was the son of Mr. and Mrs. J. R. Winford. Prior to entering Baylor University College of Medicine, Dallas, Dr. Winford attended Texas Christian University Medical Department,



DR. THOMAS E. WINFORD

Fort Worth, and later served his internship at Harris Hospital there. He spent his first year of private practice in Lone Oak and in 1922 moved to Dallas as assistant city health officer for two years; then he did private practice until 1950, with the exception of the year 1926-1927, when he did postgraduate work at the Eye, Ear, Nose, and Throat Hospital, New Orleans, La. In Dallas, the specialist served as staff member of Baylor and Parkland Hospitals; at his death he was a staff member of the Methodist Hospital, where for years he had donated his services to underprivileged children at the eye, ear, nose, and throat clinic sponsored by the Oak Cliff Lions Club. Dr. Winford was awarded a plaque by the Lions Club in 1937 in recognition of his work for underprivileged children and, in 1943, he received the Adamson Trophy which, since 1934, has been bestowed annually upon the Oak Cliff resident contributing the most outstanding work toward child welfare during the year.

Dr. Winford was a member of the State Medical Association and the American Medical Association through the Dallas County Medical Society and held membership in the American Academy of Ophthalmology and Otolaryngology, Texas Society for Ophthalmology and Otolaryngology, and the Phi Chi medical fraternity. He was a former treasurer of the Oak Cliff Kiwanis Club and a member of Knights of Pythias and the Masonic order. He was of the Baptist faith.

Surviving is his widow, the former Miss Edith Allison Emmons whom Dr. Winford married June 27, 1920, in Dallas. Other survivors include two daughters, Mrs. B. M. Menefee, Waco, and Mrs. J. A. Hughston, Dallas; a son, Pvt. Thomas E. Winford, Jr., Lackland Air Force Base, San Antonio; a sister, Mrs. I. N. Osburn, Mariana, Ark.; a brother, C. C. Winford, Long Beach, Calif.; and four grandchildren.

G. M. DECHERD, JR.

Dr. George Michael Decherd, Jr., Austin, Texas, died at his home March 5, 1951, of chronic lymphatic leukemia.

The son of the late Dr. George M. Decherd, Sr., and Mrs. Decherd, Dr. Decherd was born in Austin, December 19, 1906. He attended Austin public schools and was graduated from the University of Texas in 1926 with honors with bachelor of arts and master of arts degrees. In 1930, he was graduated with highest honors from the University of Min-

DR. GEORGE M. DECHERD, JR.

nesota Medical School, Minneapolis. After an internship and residency at the Minneapolis General Hospital, Dr. Decherd did postgraduate work on a fellowship at the University of Illinois, Chicago. He was on the faculty of the University of Texas School of Medicine, Galveston, from 1933 until his death with the exception of the years 1936 to 1938, when he served as assistant professor of internal medicine at Louisiana State University School of Medicine, New Orleans. Prior to 1946 Dr. Decherd was associate professor of internal medicine and director of the postgraduate program at the University of Texas School of Medicine. In 1946, Dr. Decherd accepted the position of director of the Student Health Center at the University's Main Branch and moved to Austin, but he was continued on the Medical Branch faculty as professor of internal medicine on leave. He played a key part in planning the new University Health Center building which was opened shortly before he died. Dr. Decherd was commissioned as an assistant surgeon of the United States Public Health Service in 1942 and served as chief of staff of Brackenridge Hospital, Austin, in 1949.

Dr. Decherd was a member of the State Medical Associa-

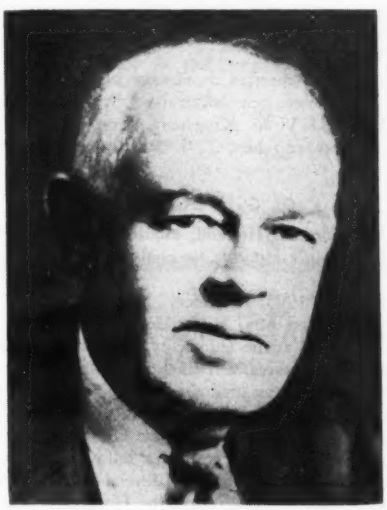
tion and the American Medical Association through the Travis County Medical Society. A fellow of the American College of Physicians, he belonged to the American Society for Clinical Investigation, American Federation for Clinical Research, Central Society for Clinical Research, and the Society for Experimental Biology and Medicine. He also was a member of the Texas Academy of Medicine, Texas Club of Internists, and the Texas and American Heart Associations. Dr. Decherd, whose field of research was experimental and clinical cardiology, was author of more than seventy scientific research papers and was listed in "American Men of Science." He held membership in Alpha Omega Alpha, honorary medical fraternity; Sigma Xi, honorary science fraternity; Phi Beta Kappa, honorary scholastic fraternity; and Phi Chi, medical fraternity. Dr. Decherd was an Episcopalian and a member of the Rotary Club.

He married Miss Helen Schultz of Minneapolis June 2, 1934. She survives with their three sons, George Michael Decherd, IV, Jonathan F. Decherd, and James William Decherd, all of Austin. Other survivors include his mother, Mrs. George M. Decherd, Sr., Brownsville, and a sister, Mrs. Dudley Williams, Columbus, Ohio.

W. J. JOHNSON

Dr. William Joshua Johnson, San Antonio, Texas, died in a local hospital March 6, 1951.

Dr. Johnson, who was born in Cookville, February 9, 1883, was the son of Clarence P. and Nettie (Pressley)



DR. W. J. JOHNSON

Johnson. He attended the Nashville Medical College, Tenn., in 1901-1902 and the Fort Worth School of Medicine until 1906. In 1907 he was graduated from the Baylor Medical College, Dallas. Specializing in mental diseases, Dr. Johnson served on the staff of the North Texas State Hospital, Terrell, until 1909; he then returned to Cookville, where he practiced privately until he accepted the superintendency of the East Texas State Hospital for the Insane, Rusk, in 1919. Four years later he reentered private practice in Tyler, but in 1924 he was appointed superintendent of the State Hospital for the Senile Insane, Austin. In 1925, Dr. Johnson accepted the superintendency of the San Antonio State Hospital, which position he resigned sixteen years later to enter private practice in San Antonio. Instrumental in introducing many advancements in the treatment of psychiatric patients, Dr. Johnson established the first beauty parlor in the state hospital system as a means of therapy. During World War II, he served as civilian psychiatrist at the Dodd Field Reception Center, San Antonio.

Dr. Johnson was a member of the State Medical Association and the American Medical Association through several county medical societies, most recently the Bexar County Medical Society. He was a diplomate of the American Board of Psychiatry and a fellow of the American Psychiatric Association. A member of the Baptist Church, Dr. Johnson also belonged to various Masonic orders including the Mystic Shrine.

Dr. Johnson is survived by his wife, the former Miss Alice Wallace of Winona, whom he married in San Antonio, April 11, 1916; two sons and a daughter, Dr. William J. Johnson, San Antonio; Dr. Robert W. Johnson, Fort Hood, and Mrs. O. B. Moon, Jr., Corpus Christi. Two sisters, Mrs. A. A. Calloway, Sweetwater, and Mrs. J. H. Coffey, Baytown, and a brother, C. P. Johnson, Waco, also survive.

F. W. HOVER

Dr. Frank William Hover, Sealy, Texas, died at his home February 13, 1951, of coronary thrombosis.

The son of George and Elizabeth (Traver) Hover, Dr. Hover was born in Germantown, N. Y., June 28, 1879. He attended Hudson High School, Hudson, N. Y., and graduated in June, 1905, from the Long Island College Hospital. After serving his internship at Bellevue Hospital, New York City, Dr. Hover settled in 1907 at Peters, Texas, where he practiced until he moved to Sealy six years later. Until his retirement in December, 1949, Dr. Hover continued his practice there and also served as local physician and surgeon for the Gulf-Colorado and Santa Fe and the Missouri-Kansas and Texas Railroads.

DR. F. W. HOVER

Dr. Hover was a member of the American Medical Association and the State Medical Association through the Austin-Waller Counties Medical Society. He was elected to honorary membership in the State Medical Association in 1950 and was a member of the Alpha Kappa Kappa medical fraternity. He was a member of the Presbyterian Church.

In 1919, the first hospital in Sealy was opened by Dr. Hover and in 1928, he built the Sealy Hospital, which he owned and operated until 1950.

In Brenham on February 19, 1908, Dr. Hover married Miss Julia Salley, who survives. Two sons, Jack Hover, Monroe, La., and Frank William Hover, Jr., Fairfield, Calif., and four grandchildren also survive as do Mrs. H. W. Murray, Woodmere, N. Y., a sister, and Byron Hover, Pittsfield, Mass., a brother.

J. A. MAYES

Dr. Joseph Allen Mayes, Denison, Texas, died in a local hospital January 16, 1951, from a heart condition.

Dr. Mayes was born in Collin County, November 24, 1878, the son of Adolph and Bernie Mayes. He attended Trinity University, San Antonio, and was graduated from the University of Texas Medical School, Galveston, in 1904. After serving an internship in Shreveport, La., Dr. Mayes began practicing in Denison in 1905 and continued active practice until he retired ten years ago. A member of the American Medical Association and State Medical Association throughout his career, Dr. Mayes was elected to honorary membership in the State Association and the Grayson County Medical Society after his retirement. He had served as president of the Rotary Club, of which he was an honorary member. Dr. Mayes was a thirty-second degree Mason and was a member of the Elks Club and Woodmen of the World. He also had served on the local school board.

Surviving is his wife, the former Miss Louise West, whom he married September 28, 1907, in Denison. Also surviving are two sons, Allen K. Mayes and Donald L. Mayes of Denison. Other survivors are two sisters, Mrs. J. C. Greer, McKinney, and Mrs. Alva Hartsfield, Dallas; a brother, Coke Mayes, Austin; and three grandchildren.

A. E. GRAHAM

Dr. Alice Elliott Graham, Corpus Christi, Texas, died at a local hospital January 18, 1951, from a coronary occlusion.

Dr. Graham was born January 1, 1908, in Pueblo, Colo., the daughter of Charles Herdman and Alice (Dickerson) Elliott. She received a bachelor of science degree from the University of Pittsburgh in 1929, a master of science degree from Johns Hopkins University, Baltimore, in 1931. Dr. Graham was graduated from the Medical College of Virginia, Richmond, in 1935. After serving an internship at the Women's Hospital, Cleveland, Ohio, she spent several months of clinical observation in Vienna, Glasgow, Paris, and London. Upon her return to the United States, Dr. Graham practiced six years in Youngstown, Ohio, where she was a staff member of St. Elizabeth's Hospital, and one year in Salem, Ohio. In 1944, she moved to Corpus Christi where she served as city-county physician until 1948 and as county physician until 1949. At her death she was a staff member of Memorial, Crippled Children's, Spohn, and Ghormley Hospitals.

A member of the American Medical Association and the State Medical Association through the Nueces County Medical Society, Dr. Graham had held memberships in the Mahoning County and Ohio State Medical Societies prior to residence in Texas. She also belonged to the Kappa Kappa Gamma sorority, Altrusa Club, and St. Patrick's Church.

On January 30, 1944, in Bethesda, Md., Dr. Graham married William Karl Graham, Kansas City, Mo., who survives. Other survivors are her mother, Mrs. Charles H. Elliott, Corpus Christi; a brother, Samuel Wilson Elliott, Cleveland, Ohio; and a sister, Mrs. James Watson Roy, Jr., Natick, Mass.

E. W. BROWN

Dr. Edna Whitcomb Brown, Freeport, Texas, died January 24, 1951, in a Freeport hospital.

Born November 22, 1873, in Jackson, Mich., Dr. Brown was the daughter of Harvey J. and Carolyn Esther (Spring) Whitcomb. She attended public school in Gilmore City, Iowa, Des Moines Teachers College, and was graduated from Hahnemann Medical College, Chicago, in 1899. Dr. Brown interned at Hahnemann Hospital and practiced medicine in Chicago for several years before returning to Gilmore City. There she and other doctors established a hospital which she managed. During World War I, Dr. Brown was resident physician for interned German women prisoners at Fort Oglethorpe, Ga. She then did postgraduate work in radiology in 1919 at Loyola Postgraduate School of Medicine, New Orleans, and practiced radiology from 1920 to 1924 at Woman's Hospital and from 1924 to 1928 at Nix Clinic, New Orleans. From 1928 until 1941 Dr. Brown was assistant radiologist at Charity Hospital of New Orleans. During World War II she was radiologist at Providence Hospital, Waco, and she had been radiologist at the Dow Chemical Company Hospital, Freeport, for five years.

Dr. Brown had been a member of the State Medical Association since 1947 through the Brazoria County Medical Society and was a member of the American Medical Association.

Dr. Brown was married to George Nathaniel Brown in Gilmore City, Iowa. She is survived by a brother, Mark H. Whitcomb, D. V. M., Kingsport, Tenn., and two sisters, Mrs. R. N. Day and Mrs. T. J. Stephenson, both of Gilmore City.

C. G. HOLLAND

Dr. Clell Gresham Holland, Houston, Texas, died February 18, 1951, in a Houston hospital after suffering a heart attack.

Born May 30, 1905, in Amory, Miss., Dr. Holland was the son of Mr. and Mrs. H. L. Holland. He received his academic education at the University of Mississippi, Oxford, from which he received a bachelor of science degree in 1925. Dr. Holland was graduated from Tulane University of Louisiana School of Medicine, New Orleans, in 1927, then interned at Mercy Hospital, New Orleans, and Shreveport Charity Hospital. He received a fellowship in surgery and urology at Cleveland Clinic, Cleveland, Ohio, and did postgraduate work at Johns Hopkins University School of Medicine and James Buchanan Brady Urological Institute, both in Baltimore. Since 1931 he had resided in Houston, where his practice was limited to surgery. Dr. Holland was one of the founders of the North Houston Hospital, for which he was chief surgeon. During World War II, he was a captain in the United States Army Medical Corps.

Dr. Holland had been a member of the American Medical Association and the State Medical Association through Harris County Medical Society since 1931. He was a member of Delta Tau Delta fraternity, the North Houston Kiwanis Club, and the Elks Lodge, Covington, Ky.

On August 4, 1945, in San Marcos he married Miss Lula Lee Neal, who survives. Other survivors are his daughters, Misses Jean Holland and Donna Lynn Holland, Houston; and a sister, Mrs. E. B. Bloodworth, Amory.